Seguin (E. C.)

LECTURES

ON THE

LOCALIZATION

OF

SPINAL AND CEREBRAL DISEASES

Delivered at the College of Physicians and Surgeons during the months of December, 1877, and January, 1878.

BY

E. C. SEGUIN, M.D.,

Clinical Professor of Diseases of the Mind and Nervous System in the College of Physicians and Surgeons, New York, etc.

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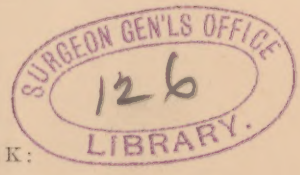

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LECTURES ON THE LOCALIZATION OF SPINAL AND CEREBRAL DISEASES.*

LECTURE I.

SUMMARY—INTRODUCTORY REMARKS—HISTORICAL CONSIDERATIONS CONCERNING LOCALIZATIONS IN THE SPINAL CORD—PHYSIOLOGICAL ANATOMY, AND PHYSIOLOGY OF THE SPINAL CORD.

GENTLEMEN:—The Faculty having again done me the honor to assign to me some of the extra Thursday Lectures this winter, I have chosen as a subject the very practical question of the Localization of Diseases in the Spinal Cord and Brain.

This topic is now engaging the attention of many of the best minds in the profession, and it is being made the object of careful observation and ardent controversy. Well-reported cases bearing on the question abound in the current medical literature.

The subject has two principal aspects. One of these, that relating to the doctrine of the localization of functions in the brain and spinal cord, is more especially interesting to the physiologist and psychologist. The other aspect, that which concerns us as practical physicians, is with reference to the possibility of making an accurate diagnosis of the seat of the lesion in organic diseases of the nervous centres.

It is this second aspect of the question which I shall discuss with you; and I shall endeavor to do it in as concise and practical a manner as possible—in such a way, in short, as shall enable you to utilize in your future daily practice the various principles and propo-

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sitions which I shall have the pleasure of presenting to you.

In other words, the following lectures will be upon the rational diagnosis of cerebral and spinal diseases.

In practice, when we have completed the examination of a patient, several questions are put to us by the patient, by his friends, or by ourselves. These are in chronological order: Is there disease? Where is the disease? What is the disease? What are we to do for the cure of the disease or for the relief of the patient? Will the patient die or recover?

Of these questions the one which our client and the world at large consider the most important, is the fourth—that relating to treatment and cure. This preference is natural, but highly unscientific; it is a manifestation of that untrained mental action which demands results and scorns methods, which welcomes empirical achievements (provided they be agreeable), and which conduces to the perpetuation of quackery of all kinds. But to the physician who is not a mere prescription-writer, who aims at infusing as much science into his practice as possible, and who believes that he is not in the world for the purpose of gratifying his patients at so much per visit, but that he owes himself a debt of training and self-culture, and who has a sincere regard for science—to such a physician the first three questions assume a justly great importance. Pray observe that I do not say paramount importance, but great importance. And the superiority of the humanitarian over the scientific duty becomes less glaring if we bear in mind the truth—and I firmly believe it to be such—that success in treatment now depends, and in the future will still more closely depend, upon the scientific study of the human subject in health and disease. In other words, I would impress you with my own conviction that the best trained and most scientific physician, if he be not a closet-student and theorizer, is the best practitioner.

We occasionally hear of an over-fine diagnosis, of extreme caution in the treatment of disease, and of the sweeping application of physiological laws to practice by men who are said to be "too scientific;" but who can number the errors, nay the sacrifices of

life which must be laid at the door of the falsely so-called "practical men," who despise learning and scientific methods? Those of us who see something of the rarer and more formidable kinds of disease fully realize that in medicine, as probably in other applicable sciences, ignorance leads to rashness and crudity in practice, while ripe knowledge conduces to success, or, at any rate, to caution in prognosis and expectancy in treatment.

Of the three diagnostic questions: Is there disease? Where is the disease? What is the disease? the second is the one which forms the key-note of these lectures. Where is the lesion producing the disordered actions or symptoms? The method to be followed in arriving at the solution of this question varies somewhat in different departments of medicine. Some lesions can be seen by the trained unaided eye, or felt by the skilled hand; the seat of others can be determined by auscultation and percussion, by the aid of instruments, such as the ophthalmoscope, laryngoscope, speculum, etc. But in the study of the nervous system greater difficulties are met with; we are, to a great extent, deprived of these physical aids; we cannot appreciate the condition of the brain and spinal cord directly by our special senses, but only by a proper interpretation of the way in which the functions of these parts are performed. In other words, the diagnosis must be made chiefly by reasoning.

What are the conditions or data necessary for correct reasoning in nervous pathology? An enumeration of these will be a brief statement of the way in which I purpose treating the questions before us.

First, you should possess a knowledge of the physiological anatomy of the parts concerned, viz., the brain and spinal cord. You are not obliged, for this purpose, to know much of the histology of the nervous tissue, but you should understand the arrangement of its various parts as recently revealed to us by perfected anatomy and embryology.

Second, you must be well versed in the mode of life and action, or physiology of the cerebro-spinal axis. You must understand, as well as the present state of science allows, what parts are excitable and

what inexcitable, which transmit impulses and sensations, which receive impressions, which originate the motor impulse, and which are endowed with special functions.

Third, you need a thorough understanding of the perverted functions of the nervous system, and of other systems connected with it—*i. e.*, of the symptoms of nervous disease; you should cultivate semeiology. And it is here more especially that previous general medical training is of great aid to the student of nervous pathology.

Fourth, you must have a clear conception of the empirical knowledge already gained by numerous post-mortem examinations of persons who have died with disease in the nervous system. You should not accept every proffered autopsy, but critically analyze before making use of it. You may demand that it shall approximate a physiological experiment in exactness and in simplicity.

Fifth, and not least, it is necessary that you have and use a keen critical and logical sense in the appreciation and combination of the above normal and morbid phenomena, in order that you may arrive at sound inductions.

The importance of the combination of the above notions for the study of nervous diseases is immense. Any hypothesis, to be acceptable, must be based upon anatomy, physiology, semeiology, and pathological anatomy. The various crude theories which have reigned awhile in medicine were such as did not fulfil this requirement: some of them were deductions from anatomical data, others applications *a priori* of physiological laws to medicine, others still based solely upon clinical studies, or upon autopsies. Indeed an effort is now being made to break down the growing doctrine of localization of lesions and functions in the brain by just such a one-sided argument. It is claimed by a high authority that facts of the fourth category (post-mortem examinations) contradict, in a perfectly overwhelming manner, the doctrine in question. Now, I trust that, however feebly I may handle the subject, I shall yet be able at the proper time to give you good reasons why we must

decline to accept and apply that authority's facts as he does.

Of the few who now deny that we can accurately localize disease in small parts of the brain during the life of the patient, I would, finally, make this critical remark. Their seemingly crushing argument is based upon what seems to me a fundamental error in the appreciation of natural phenomena, and that is, not making allowance for variability and mutability in the highly organized human frame. We now, since the labors of Darwin more particularly, admit that species in the animal and vegetable kingdoms are not fixed forms, but that they may pass into one another by almost infinitely numerous and delicately graded varieties; we know that the ultimate composition of high organic bodies (proximate principles like albumen) varies somewhat; we are prepared to occasionally find (passing to the human organism) the viscera transposed, or to meet with an exanthematous fever without its rash, with a pneumonia unaccompanied by expectoration, or with a painless peritonitis, etc.; in other words, we are, as naturalists and physicians, ready to admit variability and irregularity in the organism. Yet, in spite of all this general and special knowledge, the opponents of localization maintain that there can be no irregularity in cerebral action, and they say, by implication at least, that the decussation of the anterior pyramids must always take place, and be total. They demand of those who believe in localization that they should be able to make *every* observed case harmonize with their generalizations. Is this reasoning fit to be applied to natural history? Are we prepared to make use of the mathematical method in pathology?

I shall treat, first, of localizations in the spinal cord and medulla oblongata; and, second, of localizations in the brain proper.

I take up the spinal cord and medulla first because the phenomena are there more simple, and there is less controversy about them than there is with reference to the brain.

The now voluminous literature of diseases of the spinal cord does not teach us much with respect to the localization of its diseases. The writers of the end of the last century and of the first quarter of this—Frank, Sauvages, Rachetti, Abercrombie, Ollivier—adopted a pathological classification, which has been generally followed in systematic treatises since. Frank recognized, in a pretentious section of his great work on medicine, only spinal neuralgia (rachialgia), myelitis and spinitis (rachialgitis), and hydrorachis. Ollivier, in his last edition, 1837, enlarges greatly upon this primitive list, and describes at least eleven morbid conditions of the spinal cord and its membranes. Brown-Séquard, in 1861, besides demonstrating, as he thought, the existence of reflex paraplegia, treats of all diseases of the spinal cord briefly, and makes an attempt at localizing lesions. He admirably points out how we can diagnosticate a lesion occupying one-half of the spinal cord (hemi-paraplegia and spinal hemiplegia), and also how the height of a lesion in the cord may be estimated. Besides, he makes the first attempt at localizing disease in one of the columns of the cord, saying that when the anterior columns are alone inflamed there is paralysis without anæsthesia and little dysæsthesia. He (not knowing of Türck's researches) doubts the pathological independence of locomotor ataxia.

In 1864 appeared Jaccoud's excellent book, which chiefly treats of semeiology and ætiology of spinal diseases. He makes an advance upon previous writers, by considering the question of diagnosis of location of the lesion quite fully, and reaching the following conclusions: 1. Disease in the antero-lateral columns produces palsy without alteration of sensibility; 2. There are no symptoms clearly indicating disease of the anterior gray matter alone; 3. It is easy to recognize if the æsthesodic tract is diseased (sclerosis of the posterior columns) by pain, increased reflex,* and anæs-

* This word is used in a substantive sense, in imitation of German writers, to designate reflex movements. I think that the word is now much used orally by clinical teachers in this way.

thesia; 4. Lesion in one-half of the cord low down is indicated by hemi-paraplegia, high up by spinal hemiplegia. Leyden, writing in 1874–6, adopts the usual pathological classification, and his work is an admirable treatise. He in numerous places refers to the localization of lesions in the posterior columns, the lateral columns, the anterior cornua, the centre of the cord, and the nuclei of the medulla oblongata, pointing out the diagnosis of each. Hammond, 1876, gives a good *résumé* of the state of knowledge on the subject, basing his classification in part upon notions of localization.

But it is to monographs that we owe most in this branch of pathology.

In 1851 Türck demonstrated the extent and exact distribution of descending degeneration in the spinal cord secondary to cerebral lesions, and in 1857 he found the lesion in locomotor ataxia (then called tabes dorsalis) to be sclerosis of the posterior columns; Dr. J. Lockhart Clarke discovered the lesion of progressive muscular atrophy in 1861–2; Prévost that of infantile spinal paralysis in 1865; Prof. Charcot that of progressive labio-glosso-laryngeal paralysis in 1868; in 1865 the same observer published an autopsy of a case of sclerosis of the lateral columns (three others had been published by Türck in 1856), and in 1875 Prof. Erb, of Heidelberg, and Prof. Charcot delineated the clinical features of this disease—spasmodic tabes, or spastic spinal paralysis. In 1874 Charcot described a mixed type, in which disease of the anterior gray matter is combined with sclerosis of the antero-lateral column—amyotrophic lateral sclerosis. Disease in the central gray matter has been well studied by Hallopeau 1869–70, and Schüppel, and by Leyden, 1876. During the present year Prof. Flechsig has begun the publication of a series of papers upon the systematic diseases of the spinal cord, a work of the greatest merit, based in greater part upon the author's own pathological observations, and upon his yet more important embryological and microscopical studies upon the structure of the spinal cord (1873–6).

As regards the diagnosis of the location of non-systematic lesions (focal lesions) of the spinal cord we

have made no material advance upon the data given us by Brown-Séquard in 1861.

Let us now briefly review so much of the anatomy of the spinal cord as is indispensable to the study of the localization of its functions and lesions. I shall take it for granted that you are acquainted with the usual descriptive anatomy of the nervous centres, and call your attention chiefly to their physiological anatomy.

The spinal cord is a mass of white and gray nervous matter disposed lengthwise in columnar form, and varying in relative proportions at various points. This finer arrangement of the gray and white columns is best studied in transverse sections made at different heights in the spinal cord—for example, through its upper cervical part, through the cervical enlargement, through the lower dorsal region, through the middle of the lumbar enlargement, and near the end of the cord. In every such section we find the same parts, white and gray, but the shape of each is very different on the various surfaces. In general terms the gray substance or vesicular neurine is disposed in the centre of the section in the shape of an irregular letter H, with clubbed ends forward. These ends, anterior and posterior, are called horns; they are symmetrical on either side of one section, and the posterior reach out to the very periphery of the cord, dividing the inclosing white substance of each half of the section into two parts, the posterior column and the antero-lateral column. By means of pathological study, by histology, and more especially by means of embryology, these white columns have been much more subdivided. Following the latest arrangement by Flechsig, we find in each lateral half of a transverse section of the spinal cord the following parts, proceeding from behind forward:

1. A small triangular column of varying size, lying next to the posterior median septum, in contact with its fellow of the opposite side, the posterior median column, or column of Goll.

2. Externally to this, lying between it and the inner margin of the posterior gray horn, is a broad band containing the sensory fibres of the posterior roots, the

posterior root zone, or *zone radiculaire postérieure* (Charcot), or column of Burdach. These two constitute the posterior column of the simpler classification.

3. A small zone lying next to the periphery of the cord, just anterior to the apex of the posterior horn—the direct cerebellar column.

4. Between that and the body of the posterior horn lies an ovoid mass of fibres, the crossed pyramidal column—derived from the opposite cerebral hemisphere by way of the anterior pyramid.

5. Anteriorly to these two, occupying the sides of the section and reaching inward to the gray matter, we find the lateral columns.

6. Lying in front of the anterior gray horns, and extending forward to the periphery, is the *zone radiculaire antérieure* (Charcot), or anterior fundamental column.

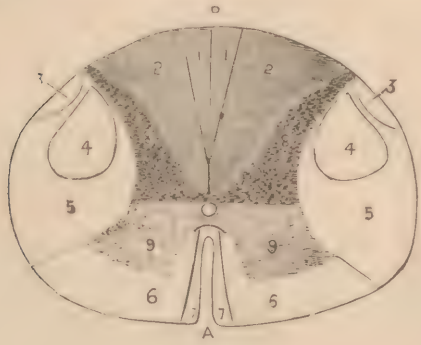

FIG. 1.

Transverse section of the spinal cord.—A. Anterior median fissure; P. Posterior median septum; 1. Columns of Goll; 2. Columns of Gerlach; 3. Direct cerebellar column; 4. Crossed pyramidal column; 5. Lateral column; 6. Anterior fundamental column; 7. Direct pyramidal column; 8. Posterior gray horns; 9. Anterior gray horns. Stippled part=gray matter. Shaded part=æsthesodic system. Unshaded part=kinesodic system.

7. A strip of white matter lying on the margin of the anterior median fissure, extending quite to its bottom, is the column of Türck, or better, the direct

pyramidal column—derived from the cerebral hemisphere of the same side, by way of the anterior pyramid.

The simple division of the gray matter into 8. The posterior horn: 9. The anterior horn, will suffice for our purpose. In the accompanying wood cut the above subdivisions are indicated by numbered spaces.

In an equally aphoristic manner allow me to recall to you the chief physiological attributes of the spinal cord:

1. It is an organ for conduction. Conduction takes place in two directions, centrifugally for motor impulses, and centripetally for sensory impressions. The paths (I will not say fibres) for sensory impressions ascend only a very small distance in the posterior columns (columns of Burdach) before they enter the gray matter, and there at once pass over to the opposite half of the cord (in man at least). Consequently we say that the conductors of sensation decussate in the spinal cord throughout its whole extent. Motor paths, on the contrary, in the spinal cord proper, remain in one-half of the organ white and gray matters; they have already decussated (in part) at the crossing of the pyramids. A strange fact to be borne in mind is that very little gray matter may suffice to transmit all sensations. The illustration (Fig. 2) represents, after Brown-Séquard, the course of sensory and motor conductors.

2. The excitability of various parts of the spinal cord is a point of some interest. It has been quite well settled that no part of the healthy spinal cord is excitable, except the posterior columns; though very lately Professor Vulpian has discovered traces of excitability in the internal part of the anterior column. However, in morbid states, a great change occurs, and even the unquestionably inexcitable gray substance becomes excitable, giving rise to various morbid sensations and to spasm.

3. The spinal cord has an autonomy of its own, giving rise to reflex motor impulses, and producing others spontaneously. It is also probable that sensory impressions are rendered more perfect in the spinal gray matter, but I am indisposed to attrib-

ute consciousness to it. This organ furthermore presides over (executes) many automatic acts, many of them highly complex; such as walking, swimming, standing, to a certain extent eating, dressing, playing upon musical intruments, etc. Part of the spinal gray matter is slowly and painfully educated to perform these actions, and can afterward do them without any marked cerebral interventions.

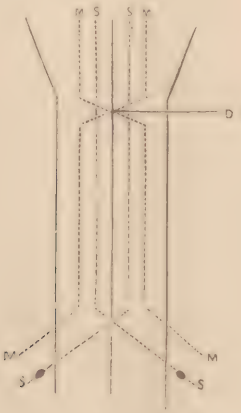

FIG. 2.

Course of motor and sensory paths in the spinal cord, after Brown-Séquard.—D, Decussation of pyramids; M. Motor paths; S. Sensory paths.

4. Besides, the spinal gray matter is supposed to possess a trophic function, to preside over the nutrition of muscles and other tissues, partly through the blood vessels, and partly directly. This question is under discussion, but the fact remains, that disease of the anterior gray matter produces marked atrophy of muscles, and may cause joint-lesions. The spinal cord is also said to embrace several so-called centres; a genital or genito-urinary centre in the lumbo-dorsal region, a cilio spinal centre in the lower cervical region,

and various subordinate vaso-motor regions; of these I recognize only one as useful in such a study as the one we are beginning, viz., the cilio-spinal centre (Budge, Waller, Brown-Séquard). There is a part of the spinal cord (in each of its halves), extending from the fifth cervical vertebra to the second dorsal vertebra, which contains vaso-motor nerve-fibres for the corresponding side of the neck, face, and eye. It also contains fibres whose normal action is to cause a dilatation of the pupil. In estimating the height of a lesion in the spinal cord a knowledge of the location of this cilio-spinal centre is of real utility.

To sum up the physiology and anatomy of the spinal cord I may divide its section-surface into two great territories, as indicated in Figure 1. The larger part, not shaded, embracing the anterior horns and all the antero-lateral columns, may be designated as the kinesodic system, and the smaller shaded portion, including the posterior horns and the posterior columns, as the æsthesodic system. As the names imply, the latter system conveys and receives sensory impressions, while the former transmits and originates motor impulses, and possibly is trophic in function.

In the next lecture I shall systematically treat of the localization of disease in these physiological and anatomical subdivisions of the spinal cord; first in the two systems, and second, in each column or horn within each system.

Lecture II.

SYNOPSIS. - 1. SYSTEMATIC LESIONS OF THE SPINAL CORD: LESIONS IN THE ÆSTHESODIC SYSTEM, LESIONS IN THE KINESODIC SYSTEM.

GENTLEMEN:—As a basis for our study of the diagnosis of the location of lesions in the spinal cord, I offer you the following classification, which I think embraces all that sound clinical observation and post-mortem examination will justify us in diagnosticating with certainty.

I. Systematic Lesions of the Spinal Cord.

II. Non-systematic or Focal Lesions of the Spinal Cord.

By the former we are to understand pathological changes which involve one of the gray or white columns of the cord for a part or the whole of its extent up and down, without extension to adjacent columns. Such lesions are almost always symmetrical in the two halves of the organ; and occasionally more than one such lesion may be present.

By the second form of lesion we understand a focus of disorganization or new tissue growth involving the spinal cord in a limited part vertically, and invading diverse columns, or even systems, transversely. These are the *lésions en foyer* of French authors.

The first group may be subdivided as follows:

I. Systematic Lesions of the Spinal Cord.

a. Lesions in the Æsthesodic System.

1. Sclerosis of the Columns of Goll.
2. Sclerosis of the Columns of Burdach.

b. Lesions in the Kinesodic System.

1. Sclerosis of the Anterior Columns.
2. Sclerosis of the Lateral Columns.
   a, with changes in Anterior Horns.
3. Degeneration of the Postero-lateral Columns.
4. Myelitis of the Anterior Horns.
5. Degeneration of Ganglion Cells of Anterior Horns.
6. Central Myelitis.

In studying the above forms of disease, I shall apply more or less rigidly the following method:

First, state the general symptoms which indicate disease in the part of the spinal cord (the system) affected. Second, accurately locate the lesion, and state the symptoms produced by it particularly. Third, say a few words concerning the disease in question.

a. Lesions in the æsthesodic system are characterized by the following symptoms: Pain, usually of a peculiar kind, hyperæsthesia, numbness, and anæsthesia; by a peculiar disorder in voluntary movements, viz.: ataxia; and, negatively, by the absence of true paralysis or spasm in the affected limbs.

1. Sclerosis of the columns of Goll, or the posterior median columns. Whether in the ascending secondary degeneration, or idiopathically produced (one case), the lesion occupies the more or less exactly triangular space lying between the columns of Burdach. At the lowest part of the cord the lesion is hardly visible, owing to the smallness of the columns at this point, but in the cervical region it is quite large and distinct. At the calamus scriptorius the lesion disappears, and so far has not been traced higher up. In Pierret's case these columns were sclerosed throughout their whole extent. In cases of ascending secondary degeneration they are affected only above the focus of disease.

The common lesion of the columns of Goll (secondary degeneration) gives rise, so far as we now know, to no symptoms; consequently, we can only infer its presence by determining the existence of a lesion capable of producing ascending and descending degeneration in the spinal cord.

The symptoms in the single case of idiopathic disease of these columns (Pierret's) are too uncertain and too badly reported to be of any use. They consisted in numbness, slight anæsthesia, and a tendency to retropulsion.

It is right to conclude that we cannot to-day directly diagnosticate disease limited to the columns of Goll.

2. Sclerosis of the columns of Burdach, or the posterior root zones. The lesion begins in the outermost portion of these columns, near the inner margin of the posterior horns, and, extending forward and inward, ultimately occupies the whole of the columns of Burdach, as shown in the accompanying diagram.

The section showing this lesion, which I now pass around, was taken from the cervical enlargement of the spinal cord of a woman who had typical locomotor ataxia in the lower and upper extremities, gastric crises, diplopia, and amblyopia. In most fully developed cases of locomotor ataxia the sclerosis is found to occupy the posterior median columns as well, *i. e.*, the whole of the posterior columns are degenerated; but, since the researches of Pierret upon the functions and pathological anatomy of the posterior median

columns, we must look upon changes in them as secondary to the sclerosis of the columns of Burdach.

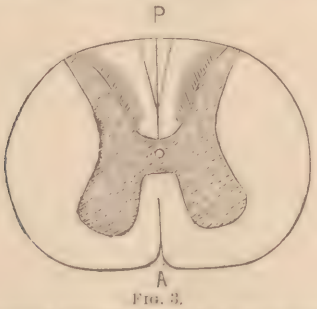

Fig. 3.

Location of essential sclerosis in locomotor ataxia.

In complicated cases of sclerosis of the posterior columns there may also be sclerosis of the lateral columns, or degenerative changes in the central and anterior gray matter.

The symptoms which are characteristic of sclerosis of the columns of Burdach are, in order of importance, pain, hyperæsthesia, anæsthesia, ataxic movements. I can only in these lectures call your attention to the great diagnostic symptoms, and must not attempt to delineate the semeiology, etc., of any disease; yet the above symptoms deserve more than a mere mention. The pains are peculiarly characteristic—nay, almost pathognomonic. They are of several varieties. First, sharp, lancinating cutaneous pains, appearing in spots, usually circular or oval, in size from one to two inches in diameter, in any part of the limbs which are afterward to become ataxic. A few shootings may take place in one spot, or it may be the seat of pain for twelve or twenty-four hours. A capital point is the capriciousness with which the pains appear in any and all parts of the limbs—toes, thighs, calves, shins, etc. They are vagrant or vagabond pains, in sharp contrast to the fixed *neural* pain of neuralgia. Second, deep, boring pains; and third, tearing or bruising deeper pains, affecting the articulations as well.

These pains are also vagabond and capricious in their mode of appearance. They may exist for years without anæsthesia and ataxia showing themselves, and both patient and physician are apt to speak of the pains as "rheumatic," instead of as pathognomonic of an incurable disease. Hyperæsthesia accompanies and succeeds the pain, more especially in the patches of skin which are the seat of stabbing pains. During the paroxysm, and for hours afterward, the patch or patches are exquisitely sensitive, and hyperalgesia exists. Anæsthesia is said to be present very early in the disease, as well as later. In the former case it occupies patches of skin on the legs, arms, and trunk, according to the extension of the disease. In the second case it is found under the feet, later in the legs, and may involve the whole of the lower (and upper) extremities toward the close of the disease.

The ataxic walk consists in a jerky, stamping gait, the abductors and extensors acting over forcibly; the want of co-ordination affects large muscular groups, thus differing from chorea, paralysis agitans, etc. But a somewhat similar ataxia may be present in cases of intra-cranial disease, so that I would urge you to fall back upon the sensory symptoms for your diagnosis.

Many practitioners employ what they consider a sure and ready test for locomotor ataxia, viz., they bid the patient try to stand with his eyes closed. If he stagger or fall, he is said to have the disease. Now, I would have you all bear in mind that this test is worthless. Oscillating with closed eyes is a symptom common to many cerebral, spinal, and peripheral morbid states, as hysteria, myelitis, etc. I once artificially froze the soles of a patient's feet, and showed him in this amphitheatre with an excellent stagger when his eyes were closed. He staggered because his feet were anæsthetic, and that is one reason why hysterical women and the bearers of myelitis do the same. We may safely put it in this way: patients with locomotor ataxia, at a certain stage, do stagger when their eyes are closed; but, as the bearers of several other spinal diseases do the same, the sign has no special value.

For many years, and even long after the patient is

absolutely unable to stand, there is almost no paresis and no diminution of reflex or of electrical excitability in the affected extremities, unless the sclerosis extend forward into the gray matter.

We ought, I think, to be able to correctly diagnosticate this lesion in its first stage, viz., when only pain and hyperæsthesia are present.

Historical Considerations—The clinical history of locomotor ataxia has been fairly well known since 1835 or 1840; German physicians describing it in great part under the name of Tabes Dorsualis. Romberg, in 1853 (and earlier), gave almost a perfect picture of the disease; but it was reserved for Duchenne (1859–60) to recognize the value of the pains and of the ataxia, as distinguished from paresis, as cardinal symptoms. Although Duchenne's description of the affection has hardly been improved upon, it must be admitted that he knew nothing of its pathological anatomy, and was quite wrong in his explanation of the symptoms. Yet the lesion of locomotor ataxia, or tabes dorsualis, had already been discovered by Türck in 1857. Unfortunately, he buried his remarkable paper in the Transactions of the Academy of Vienna; and Gull (Sir William) independently made the same discovery in 1859. In France, Bourdon worked out the pathological anatomy in 1861, and since, our knowledge upon the point has grown enormously. The anomalies of locomotor ataxia, both semeiological and pathological, have been best studied by Charcot and his pupils. In 1873, Charcot and Pierret published their cases demonstrating that the primary essential lesion of locomotor ataxia involves the columns of Burdach.

b. Lesions in the kinesodic system are characterized by the following symptoms: paresis or paralysis, spasm, and muscular atrophy; and, negatively, by the absence of anæsthesia, or of marked and permanent pain or numbness.

1. Sclerosis of the true anterior columns or columns of Türck, or, best, the direct pyramidal fasciculi, has been known pathologically since the publication of Türck's first papers in 1851. Its semeiology is, however, unknown. We can infer its existence in those

cases in which we diagnosticate lesion No. 3 (*infra*), because pathological anatomy shows us that the two lesions are usually simultaneous.

2. Sclerosis of the lateral columns. The lesion consists in an increase of the neuroglia and atrophy of nerve-fibres in the lateral masses of white substance throughout a greater part or the whole of the spinal cord. It may exist alone as a primary lesion, or may be associated with other (secondary) morbid processes, such as No. 4 (*infra*).

The symptoms consist in progressive paresis of the lower limbs, and later of the upper, with increased reflex, and a tetanoid state of the extremities. There is very little sensory disturbance, never anæsthesia; the paralyzed and tetanized muscles do not undergo atrophy; and the bladder is not in the true sense of the word paralyzed.

These symptoms have been designated by various names. Erb calls the symptom-group spastic spinal paralysis; Charcot, spasmodic tabes; and I would suggest the term tetanoid paraplegia or paralysis. Erb in 1875, and Charcot in 1876, Erb again this year, have fully described the clinical aspects of the disease.

In 1873 I described, under the title of tetanoid pseudo-paraplegia, the symptoms of the semi-developed disease, of that transition period when the patient is still able to get about upon his morbidly stiffened limbs. I failed, however, to seize upon the whole clinical picture.

The pathological anatomy of this affection does not as yet rest upon a firm basis. Türck in 1859 published three autopsies in cases of this sort, but their clinical features had not been worked out. In 1865 Charcot reported sclerosis of the lateral columns as the lesion found in a woman who had suffered from aggravated hysteria, with contractures of the extremities, for many years. In the essays of 1876-7 no other autopsies are recorded.

2 *a*. Combination of lesion of the anterior horns with sclerosis of the lateral columns. Charcot, in 1874, first called attention to this complex systematic

lesion, and designated the disease as amyotrophic lateral sclerosis.

The cervical enlargement of the spinal cord being nearly always the first part affected, we observe that the symptoms appear first in the hands; a paralytic atrophy setting in with considerable rapidity. Formication and fibrillary movements may be present. The atrophy resembles more that observed in myelitis of the anterior horns (No. 4) than in degeneration of the ganglion cells of the anterior horns (No. 5). In a short time a degree of rigidity appears in the upper extremities, and the legs become first paretic, later, rigid and contractured. This contracture may be greatly relaxed while the patient is in bed, but is exaggerated if he try to stand or walk. In consequence of great atrophy of the interossei, the "claw-hand" deformity may appear. As a rule, the muscles of the lower limbs do not waste. The bladder and rectum are not paralyzed, and no anæsthesia is observed. If the patient's life be prolonged, the disease invades the nuclei of the motor bulbar nerves, and to the above picture we have superadded the symptoms of labio-glosso-laryngeal paralysis.

Cases of this kind had been observed prior to 1874, but it is Charcot who in that year first gave us a clear statement of the pathological anatomy and semeiology of the disease. The connection between the two lesions, disease in the anterior horns, and sclerosis of the lateral columns, is by no means understood, and it may be questioned whether they are pathologically related.*

3. Degeneration of the posterior part of the lateral column, or (better) of the crossed pyramidal fasciculus. The lesion occupies, in a transverse section, a

* Since this lecture was delivered, Prof. Flechsig, of Leipzig (in *Archiv der Heilkunde*, 1878, Heft 1), has made an elaborate critique of Charcot's cases and others, and claims that the lesion in the lateral columns is chiefly in the postero-lateral columns, as in descending degeneration from cerebral lesion, and that the nature of the lesion seems more like a degenerative than a sclerotic one. Flechsig suggests that future research may reveal a double lesion in the cerebrum, or, at any rate, in the upper part of the intra-cranial motor tract. This view seems to me well worthy of consideration, and a careful attempt should be made to verify it.

part of the white substance which lies between the lateral column and the posterior gray horn. It is separated from the periphery of the cord by healthy tissue, the direct cerebellar fasciculus. This alteration of tissue is secondary to a lesion in parts of the nervous centres above the decussation of the pyramids; in the anterior pyramids, the great motor tract in the basis cruris cerebri, the anterior part of the internal capsule, the nucleus caudatus, the convolutions comprising the excitable districts of the cerebrum. Lesions of any of these parts cause what is known as secondary degeneration throughout the motor tract, to the lowest part of the spinal cord. Consequently, the lesion in the spinal cord is always (?) on the side opposite to that on which the primary, supra-spinal disease exists. Hence, also, we usually find descending degeneration only in one-half of the cord. It should be remembered, however, that, as a portion of the anterior pyramid of the medulla does not decussate, but descends as the direct pyramidal fasciculus, or column of Türck, we must expect to find in many cases a similar lesion of this fasciculus on the same side as the supra-spinal lesion (No. 1). For example, after a lesion involving the great motor tract in the left hemisphere, we shall find descending degeneration of the crossed pyramidal fasciculus in the right half of the cord, and of the anterior column, or direct pyramidal fasciculus in its left half.

The symptom characteristic of this morbid condition is the secondary contracture, or late contracture, which so often succeeds attacks of hemiplegia, being superadded to paralysis or anæsthesia. This lesion may be complicated with No. 4, when atrophy of some of the paralyzed and rigid muscles supervenes.

In briefly mentioning the historical data connected with this lesion, I must again mention Türck, the great pioneer in the pathology of systematic lesions of the spinal cord. In 1851 and 1853 he exactly described the crossed part of the lesion. An excellent study of this and other forms of secondary degeneration was made by Bouchard in 1866, under the direction of Profs. Vulpian and Charcot. Last year Flechsig published his remarkable researches upon the ner-

vous centres, and more exactly defined the seat of both the crossed and the direct degenerations. To Prof. Bouchard is due the credit of completing the clinical picture, by pointing out the value of the symptom contracture.

Lecture III.

1. SYSTEMATIC DISEASES OF THE SPINAL CORD, CONTINUED; LESIONS IN THE KINESODIC SYSTEM. 2. NON-SYSTEMATIC OR FOCAL LESIONS OF THE SPINAL CORD; LESIONS AT DIFFERENT HEIGHTS IN THE ORGAN.

GENTLEMEN:—There remain for consideration a few of the systematic lesions of the spinal cord.

4. Myelitis of the anterior horns, or poliomyelitis anterior. Like all affections of the kinesodic tract, this affection is characterized by the predominance of motor symptoms and the absence of sensory ones. But we also meet with great trophic changes—muscular atrophy—in this disease.

The lesion consists, as we know from the autopsies by Gombault, by Cornil and Lépine, and by Déjérine, in an inflammation of the anterior gray horns of the spinal cord, leading to atrophy, and even destruction of the motor (and trophic?) ganglion-cells. The change in the cells is acute pigmentary degeneration.

In those regions of anterior gray matter corresponding to the paralyzed parts, hardly any motor ganglion-cells remain. Other lesions are degenerative changes in the motor nerves, as far as their termination, and muscular atrophy, usually without fatty metamorphosis.

The symptoms of the affection vary in the three varieties: acute, sub-acute, and chronic.

a. In acute febrile poliomyelitis anterior we observe a sharp, remittent or continued fever, lasting one or several days, accompanied in many cases by pains in the limbs, and sometimes by slight numbness. This is followed, suddenly, as a rule, by extensive paralysis, the fever ceasing. The paralysis may affect all the

limbs, or two of them, or one only; it tends to diminish spontaneously to a marked extent. No anæsthesia is present, and but little numbness. Reflex movements are reduced or abolished, the bladder and rectum act normally, and there is no tendency to bed-sore. But other, even more characteristic symptoms, soon follow. In a few days the nerve-trunks in the severely palsied limbs lose their galvanic and faradic excitability, and the muscles, while ceasing to respond to the faradic current, contract slowly, and with abnormal formula, to the galvanic current—we have the degeneration-reaction. A little later, after two or three weeks, the palsied muscles undergo rapid atrophy, an atrophy which is progressive if no recovery is to take place. Ultimately only one muscular group may remain paralyzed and atrophied.

b. There is a non-febrile acute myelitis anterior. The patient, usually a child, is put to bed well, and awakes in the morning with one or more paralyzed limbs, with the subsequent symptoms as above.

In these two forms are to be ranged nearly all cases of infantile spinal paralysis so-called, and many cases of the same disease occurring in the adult.

c, d. Febrile and non-febrile subacute myelitis of the anterior horns differ from the above only in degree of acuteness and in rapidity of development, and need no detailed description.

e. Chronic myelitis of the anterior horns is often mistaken for progressive muscular atrophy; yet a diagnosis, is, I think, frequently possible. Often in this variety of poliomyelitis there occur severe neuralgic pains in the limbs which are to undergo palsy and wasting. These phenomena, when they appear, weeks or months after the first symptoms, attack whole muscular groups at once, and we do not observe the fibrillary or fascicular wasting of progressive muscular atrophy. The reactions are like those found in the acute and subacute form. No anæsthesia appears. This rare form has been observed in children and in adults.

It is only very recently that we have had a correct knowledge, clinical and pathological, of myelitis anterior. Prior to 1865, infantile spinal paralysis, though well known clinically (Heine, 1840), was

thought to be due to congestion of the spinal cord, etc.; but in that year Prévost, working with Charcot, discovered the lesions in the anterior horns, and since numerous autopsies have yielded the same results. As regards the disease in the adult, it was correctly observed and classified as far back as 1847 (Duchenne), and in subsequent years by Charcot and others. Its pathological anatomy was not discovered until 1873 (Gombault) and 1875 (Cornil and Lépine). Although we need more light upon the intimate nature of the pathological changes occurring in this disease, I believe it to be now quite firmly established in nosology. Its diagnosis should be readily made by all practitioners.

5. Degeneration of the ganglion-cells of the anterior horns. As indicated by the name, the lesion in this affection is degenerative rather than inflammatory. This is true in the sense that changes in the neuroglia are wanting as a rule, and that the molecular death of the ganglion-cells takes place very slowly. This is in marked contrast to the suddenness and extent of the lesion in No. 4. Ganglion-cells are found in every stage of transition from simple increase of normal granular contents to mere roundish masses of granules—granular bodies; in some parts of the anterior horns not even vestiges of cells remain.

The symptoms of this lesion are fibrillary contractions followed by atrophy. Sensory symptoms are wholly wanting as a rule, and in a few cases the wasting limbs are the seat of some neuralgic pains. There is at no time a true paralytic condition, since the loss of power is precisely in proportion to the destruction of muscular tissue.

The mode of occurrence of the atrophy deserves a remark. Whole muscles or muscular groups do not waste away rapidly as in No. 4, but the muscular tissue undergoes change, bundle by bundle, very slowly. It thus happens that we see one or two large fasciculi in a muscle quite atrophied, while the adjacent fasciculi of the same muscle are normal, or only show fibrillary contraction. Several months may elapse before a muscle be wholly atrophied. Another feature of the atrophy is that it attacks by preference certain

muscular groups, as those of the hand, chest, thighs, etc. It also affects simultaneously, or nearly so, parts which are symmetrical and homologous. If we examine the wasting muscles with the faradic current, we obtain yet another diagnostic sign: in this affection reaction to the faradic current is lost only in the absolutely atrophied muscles or parts of muscles. This loss of reaction is in direct proportion to the atrophy, whereas, in myelitis of the anterior horns whole muscles and muscular groups lose their faradic reaction *en masse*, and this, too, often before any marked degree of wasting has appeared. By the latter test, by the distribution of paralysis and atrophy, and by the predominance of neuralgic pains in chronic myelitis of the anterior horns, we may nearly always distinguish it from progressive muscular atrophy. Of course, the two diseases are congeners, and their pathological relationship may even be closer than we now suspect.

Cruveilhier, Aran, Duchenne, and Roberts admirably described the clinical aspects of this disease, and its naked-eye pathological anatomy. But it is to the very recent labors of Lockhart Clarke (1861-2), and of Charcot and his pupils, that we owe the exact determination of the lesion in the anterior horns of the spinal cord. It is also only within the last ten years that we have clearly distinguished pure muscular atrophy from the various forms of symptomatic atrophy.

6. Central myelitis. An inflammation of the central parts of the spinal gray matter, involving the æsthodic and kinesodic tracts, extending in some cases throughout the whole length of the organ. The proliferative changes and exudations result either in the formation of a central plug, or the development of a cavity, which is filled with clear fluid. The anterior horns and the various columns of the spinal cord are more or less involved through extension of inflammatory action, or by compression. As might be inferred from the above, the symptomatology of the affection is obscure and complex. Early in the disease, disorders of sensibility—as numbness, formication, and pain, followed by anæsthesia—are prominent. Irregularly distributed paralysis, with or without atrophy, is also

present. The diagnosis in this early stage, which may last years, is next to impossible. When the disease is fully formed, we have quite a distinct symptom-group. The arms alone are sometimes paralyzed, atrophied, and anæsthetic, while the legs are the seat of abnormal reflex, even to the degree of tetanoid walk. In other cases we see a general paralysis and universal anæsthesia, with contracture of some muscular groups, paralysis of the bladder, and the appearance of bed-sores. If the cervical enlargement be the seat of diffused central myelitis, pupillary symptoms are seldom wanting, and the pulse is accelerated. The disease is eminently a chronic one, years being required for its full development.

Historical considerations.—Ollivier (1836) observed and described central myelitis with formation of cavities, but not much was learned of the disease and its semeiology until Schüppel published his paper (Ueber Hydromyelus) in 1865. Hallopeau, in 1871–2, contributed a series of elaborate articles upon the subject to the *Archives Générales de Médecine*, and in the last few years Westphal and Leyden have paid considerable attention to this rather rare form of disease.

2.—NON-SYSTEMATIC OR FOCAL LESIONS OF THE SPINAL CORD; LESIONS AT DIFFERENT HEIGHTS IN THE ORGAN.

A variety of lesions may involve the whole or a large part of the spinal cord at a given level, extending transversely through its various columns. These are focal lesions, and chief among them we find: injuries of all kinds; compression by bone or by a tumor; transverse sclerosis; transverse softening; hemorrhage in cord; tumor in the cord, etc.

The nature of the lesion is sometimes such (fracture of vertebræ) as to indicate at once the seat of injury to the spinal cord; but in many cases the peculiarity in the symptoms is not due to the nature of the lesion, but to its location high up or low down. The diagnosis of this location is possible only by the aid of anatomical and physiological knowledge.

The following diagram, made from data furnished

by Malgaigne, may assist in estimating the height of a lesion. It indicates the point of origin of the important nerves and plexuses, and the seat of so-called centres relatively to the spinous processes of the vertebræ.

In general terms, we may say, that these focal lesions give rise to paralysis, numbness and anæsthesia, to modifications of the reflex function, usually an increase; that they cause paresis or paralysis of the bladder and the sphincter ani, and that they set up a great liability to bed-sores. This general sketch varies greatly from that of the symptoms of any of the systematic spinal lesions, except No. 6, central myelitis. This great dissemblance is a necessary result of the difference in the location of lesions; focal lesions involving kinesodic, æsthesodic, and trophic parts of the cord, and cutting off the inhibitory action of the encephalon (Setschenow), upon the spinal cord.

I shall attempt to make clear to you the diagnosis of lesions placed (1) in the lower lumbar enlargement; (2) just above the lumbar enlargement; (3) in the mid-dorsal region; (4) in the cervical enlargement, and (5) in the upper cervical region.

1. Focal, transverse lesions in the lower part of the lumbar enlargement. The motor symptoms produced by such a lesion are paralysis of the muscles innervated by the sciatic nerve—those of the feet, legs, posterior aspect of the thigh and the nates. The sphincter ani will be weak or paralyzed; the bladder unaffected. The reflex movements of the paralyzed muscles are reduced in force, or absolutely wanting; wanting if the whole of the gray matter in the lower end of the cord is diseased in such a way as to destroy its functions. Dependent also upon this extent of lesion downward, is the state of muscular irritability. If the condition be as just described, the paralyzed muscles undergo atrophy, and lose their faradic contractility. The sensory symptoms are various; at the beginning of the disease (often before paresis) there is numbness in the soles of the feet, without anæsthesia. Later the numbness may appear in the whole foot, the calf, the posterior aspect of the thigh, and actual anæsthesia may supervene. If the soles of

the feet lose their sensibility, the patient is no longer able to maintain his equilibrium when his eyes are

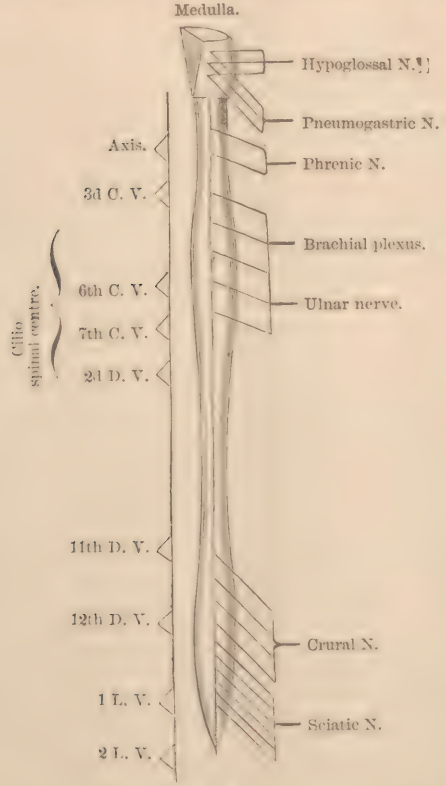

FIG. 4.

Relation of spinous processes of vertebræ to spinal nerves. After Malgaigne, *Traité d'Anatomie Chirugicale*, vol. ii., pp. 32–3.

closed. Another sensory symptom of importance is the cincture or band feeling, which, whether like a cord

or like a belt round about the palsied parts, indicates (in accordance with the distribution of sensory nerves) the upper limit of the spinal lesion. Thus, in the condition we now study, the cincture feeling will not be about the waist or groin, but around the ankle, leg, or thigh. The above symptoms make up an incomplete paraplegia.

2. Focal, transverse lesions, situated just above the lumbar enlargement. Conduction to and from the brain is interfered with, but the gray matter of the lumbar enlargement retains its functional activity in great measure; hence we have a very different semeiology from that described above. In addition to weakness in the feet, there is developed a more or less complete paralysis of the whole of the lower extremities, from the pubis down. The rectum and bladder are paralyzed, the latter showing defective action by slow, interrupted micturition, or by retention, while constipation expresses the rectal paresis. The reflex movements of the paralyzed limbs are usually exaggerated, sometimes enormously so. This increase in reflex movements gives rise to the combined tonic and clonic movements of the paralyzed limbs, which Brown-Séquard years ago designated spinal epilepsy. This often seems spontaneous, when the patient is in bed for example, but it is certain that peripheral irritations, whether from the contact of the bed-clothing or from feces in the rectum, urine in the bladder, etc., always precede and cause it. The preservation and increase of reflex is owing to the continued (increased) activity of the lumbar gray matter below the lesion, and the cessation of cerebral inhibitory action. Dependent upon the former fact also, we observe that the paralyzed muscles do not undergo positive atrophy, and that their electrical reactions are normal or exaggerated. Erections occur, and coition may be accomplished in these cases and in those to be described in the next paragraph.

As regards sensory symptoms we have, as in No. 1, numbness and anæsthesia in the paralyzed parts, extending as high upward as the groins or waist. The cincture feeling is nearly always present, and is placed by the patients round about the body at the level of

the hips, or waist just below the umbilicus. In some cases the cincture is incomplete, and the feeling is likened to a firm grip in the patient's side or hip. The above description is a picture of common, complete paraplegia.

3. Focal, transverse lesions in the middle or upper dorsal region. The motor and sensory symptoms produced by a lesion so placed are very similar to those just described, with the following additions. The abnormal reflex movements are often more marked than in No. 2, and the cincture feeling, index of the upper limit of the lesion, is placed at or above the umbilicus, around the lower ribs, or even just under the arms. In this condition the rectum and bladder may after a while partly regain their functions; *i. e.*, their contents are involuntarily expelled from time to time by reflex action.

If the urine dribbles away it is not because the "sphincter" (?) of the bladder is paralyzed, but because there is retention and overflow—a state demanding the daily use of absolutely perfect and carbolized soft catheters. In the earlier stages of the affection, the occurrence of increased reflex action in the paretic limbs gives rise to a tetanoid state during attempts at standing or walking, and too hasty, quasi-involuntarily micturition and defecation. Later in the disease the paralyzed limbs may become contractured, by reason of secondary changes in the lateral columns.

4. Focal, transverse lesions in the cervical enlargement. According as the lesion suddenly or gradually affects the whole of the cord transversely, or according as it is placed on the lower or upper portions of the enlargement, somewhat different symptoms are obtained. They always, however, bear a general resemblance to those in Nos. 2 and 3.

a. A partial lesion may for a time produce symptoms, numbness and paresis, in the arms and hands alone, the lower limbs being only weak and showing increased reflex. Later, as the lesion extends, the legs as well as the arms are paralyzed, and the cincture feeling exists high up.

b. A lesion involving the cord at the level of the eighth cervical and first dorsal nerves (see sketch) will

give rise to paralysis, often with atrophy and loss of faradic reaction in those muscles of the upper extremities which are animated by the ulnar nerves, *i. e.*, nearly all the small muscles of the hands, and some of the flexors of the wrist and fingers. There will be sensory symptoms in the same district; and the cincture feeling, if present, will be across the upper part of the chest. The lower extremities are paretic or wholly paralyzed, numb or anæsthetic, according to the completeness of the destruction of the spinal tissue. In severe cases nearly all the intercostal muscles will also be paralyzed, and thus life will be much more jeopardized than by lesions placed lower down. The danger is all the greater because the expiratory muscles (intercostals, triangularis sterni, abdominal muscles) are paralyzed also.

c. If the lesion be situated in the upper part of the enlargement, the motor and sensory symptoms will be apparent in nearly the whole of the upper extremities, as well as below them. The reflex capacity, the state of bladder and rectum, the faradic reactions of muscles remain substantially as in Nos. 2 and 3. The cincture feeling is referred to the level of the clavicles, or a little lower, across the chest and the arms below the deltoid. The difficulty of breathing is even greater than in *b*. The symptom-groups produced by lesion No. 4 are often designated by the names of cervical paraplegia, or general paralysis.

In case of lesion in any part of this region (from the level of the fourth dorsal to that of the fourth cervical nerves) there may be pupillary and facial vaso-motor symptoms. If the lesion be of such a nature as to cause irritation of the cilio-spinal centre, the symptoms are dilatation of the pupils and pallor of the face, while if there be a loss of the activity of the cilio-spinal centre, the pupils are small and the face and ears flushed and hot. It must be added that these vaso-motor and ciliary symptoms are not by any means as frequently observed as theory and experimentation would lead us to expect.

The same may be said with reference to some peculiarities in the action of the heart and variations in the bodily temperature which have been observed. In

severe traumatic lesions in this region, we often find retardation of the pulse, and great elevation of the temperature of paralyzed parts.

. Focal, transverse lesions in the upper cervical region. These, like No. 4, produce cervical paraplegia, but a much more complete one. The patient is wholly paralyzed below the head, and the entire body may be anæsthetic. Of necessity the phrenic nerves are paretic or paralyzed, according to the completeness of the injury to the cord, and life is almost immediately terminated by asphyxia. Cases of this category are nearly always of a surgical character; non-traumatic lesions of this region being exceedingly rare. There may be ciliary and facial vaso-motor symptoms here as in No. 4, and the bodily temperature and pulse-rate are variable. Life is preserved too short a time to allow of much study of these symptoms. In slowly developed lesions we may have phenomena of irritation, as hiccough, dyspnœa, acceleration of the pulse, together with paretic symptoms in the arms and chest, later in the legs.

Lecture IV.

2. Non-systematic or focal lesions of the spinal cord continued; lesions involving one lateral half of the spinal cord, in its lower and upper regions; diagnosis of spinal hemiplegia. Anatomy and diseases of the medulla oblongata.

Gentlemen:—When a focal lesion, caused by spontaneous disease, or by traumatism, involves one lateral half of the spinal cord more or less exactly, we observe striking and characteristic symptoms in the patient. Some of these are in relation to the height of the lesion in the organ as pointed out in the preceding lecture, but the most important ones are dependent upon the fact that one lateral half of the cord is injured in its kinesodic and æsthesodic tracts. A reference to Figures 2 and 4 in preceding lectures will facilitate the comprehension of what I shall have to say upon these lesions.

According as the lateral focal lesion is placed low down or high up in the spinal cord, we denominate the symptom-groups as hemi-paraplegia in the first case, spinal hemiplegia in the second.

a. Hemi-paraplegia. A tumor compressing one lateral half of the spinal cord in its dorsal (case by Charcot) or lumbar regions, a knife-cut, a contusion by a piece of broken vertebra, or a patch of hemorrhage or softening, will give rise to this symptom-group. Let us suppose the lesion to be situated in the right half of the spinal cord. As a result (see Fig. 2) the motor paths from the brain and upper spinal cord to the right lower extremity are cut off, together with the sensory paths which, crossing the median line below the lesion, supply the left lower extremity with sensibility.

In the living human subject we observe motor paralysis, more or less complete in the right lower extremity, and the sensibility of this member is preserved or increased. If the lesion be traumatic and quite complete, hyperæsthesia and increased temperature are present. In the left lower extremity, on the contrary, we find no paralysis, but more or less complete anæsthesia. It is noteworthy that the so-called muscular sense is not abolished in that anæsthetic limb. The bladder and rectum may be paralyzed. In some cases the distribution of symptoms in the two extremities is not as typical and clear as above stated, some paresis appearing on the side opposite the lesion, and slight loss of sensibility existing on the paralyzed side. This, I need hardly say, is owing to the fact that the lesion crosses the median line.

As regards constriction bands, increased reflex, nutrition of muscles, and visceral paralysis, regard must be had to the exact height of the lesion in the cord, as determined in sections 1, 2, 3 of the preceding lecture.

This form of paraplegia can be exactly reproduced in animals. It is now nearly thirty years since Brown Séquard showed to the Biological Society of Paris, animals (Guinea pigs, etc.), in whom hemi-paraplegia had been produced by cutting one lateral half of the cord in the dorsal region. The operation

is not very difficult, and the results are always striking.

b. Spinal hemiplegia. A lateral focal lesion in the cervical enlargement, or above it, will give rise to the following symptoms, more or less exactly distributed. If the right half of the organ is the seat of the lesion, we observe that the arm and leg on the same side are paralyzed, the intercostals usually escaping, while on the opposite side there is no paralysis, but more or less perfect anæsthesia exists to the median line of the body, and as high as the limit of distribution of sensory nerves coming from the spinal cord just below the lesion. The absolute height of the lesion is to be determined as in sections 4 and 5 of Lecture III. In these cases we nearly always find, on the same side as the injury, contraction of the pupil, redness and increased temperature of the face and ear. These symptoms are due to paralysis of the vaso-motor and ciliary centres in the spinal cord. Often the paralyzed limbs are hotter than the anæsthetic.

In some cases, owing to incomplete destruction of one lateral half of the cord, the arms, thorax, and face alone exhibit symptoms, the legs remaining normal. In other cases various degrees of abnormal reflex action are present in the affected lower limbs; sometimes more on the anæsthetic side.

We owe a clear conception of these interesting forms of spinal paralysis to Brown-Séquard, although some cases of spinal hemiplegia had been placed on record before him (Sir Chas. Bell, Ollivier, Oré, and others). This distinguished physiologist and physician produced spinal hemiplegia and hemi-paraplegia in animals from 1849 on, and in 1863-5 published elaborate memoirs, in which he collected all the cases then known, and critically studied their semeiology. In 1868 and 1869 the same author published other cases and remarks which have placed our knowledge of this subject upon a firm foundation. Some doubt exists as to the absolute correctness of Brown-Séquard's law of the course of motor and sensory paths in the spinal cord in animals, but in man it would seem, from the study of many cases, that the law holds good.

ANATOMY AND DISEASES OF THE MEDULLA OBLONGATA.

Leaving the diseases of the spinal cord to proceed to the consideration of those of the medulla oblongata, I must again refresh your memories upon the physiological anatomy of the parts concerned; mentioning only such data as will be of use to us in our diagnosis of location of disease. The first, and perhaps the most important point I wish to call your attention to is the decussation of the anterior pyramids at the junction of the spinal cord and medulla. This decussation is made up of bundles of nerve fibres coming from the anterior pyramids of the medulla, crossing the median line, and continuing their way down the cord in each of its halves, constituting that part of the lateral column known as the crossed pyramidal column. Another bundle extends downward, without crossing the median line, into the inner part of the anterior column, the so called direct pyramidal column (compare Fig. 1., Lecture I.). According to the books, the uncrossed portion of the pyramidal fasciculus, the direct pyramidal column, is much smaller and less important than the other bundle of fibres which cross the median line.

Of immense importance for the study of localizations in the brain, and particularly for the estimation of the symptom hemiplegia, is a knowledge of the exact extent and regularity of this decussation, or more strictly speaking, semi decussation. If we are to follow text-books we shall be led to believe that the semi decussation always occurs in about the same proportion on the two sides: a view which Brown-Séquard and others admit while trying to demonstrate that a lesion of the brain may produce paralysis on the same side of the body. Still, in past times a few facts had been recorded against the constancy of the pyramidal decussation, but it was not until Flechsig published his embryological researches in 1876, that it was known how *very variable* is this decussation. Examining the spinal cord of sixty fœtuses, this investigator found that the proportion in the size of the crossed and direct pyramidal columns varied almost infinitely between the following figures: in one case

the direct pyramidal column was equal to 90, the crossed column to 10. In another case at the other extreme the proportion was 0 to 100; in other words all the pyramidal fibres had crossed the median line. There was no case in which there was absolutely no decussation, but Flechsig correctly remarks that there is no reason why such a specimen should not be met

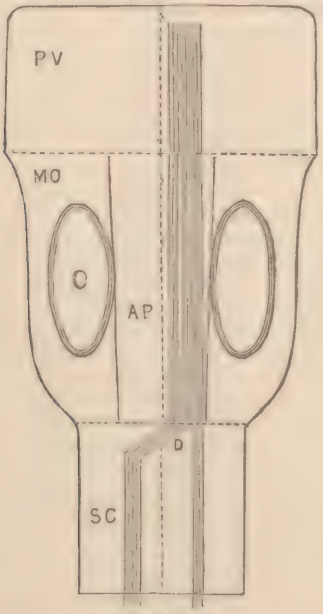

FIG. 5.

Diagram explicative of pyramidal decussation: P.V. pons Varolii M.O. medulla oblongata; O. olivary body; A.P. anterior pyramid; D. decussation; S.C. spinal cord. The direct and crossed bundles vary very much in size, as shown, in the following ratios of crossed and direct: 100:0, 92:8, 84:16, 70:30, 52:48, 35:65, 10:90. (*Flechsig.*)

with. The majority of the fœtuses had an ordinary semi-decussation, the crossed bundle predominating over the direct. The above variations in the pyramidal decussation are diagrammatically represented in Fig. 5.

Besides, Flechsig determined that there were other irregularities in the proportionate size of the four columns (two crossed pyramidal columns, two direct) which result from the breaking up of the pyramids, but this is not essential to our present study.

The practical bearing of this discovery is very great, for it will be readily understood that in the exceptional cases in which the pyramidal decussation is nearly wanting, an unilateral lesion of the medulla, pons, or brain must give rise to symptoms of paralysis on the same side as the lesion.

Again, in those few cases in which the semi-decussation is nearly equal, an unilateral lesion above the spinal cord will produce weakness on both sides of the body. Lastly, in the immense majority of cases, those in which most of the pyramidal fibres cross the median line, we obtain the classical crossed hemiplegia. It is ignorance of these researches of Flechsig which makes that distinguished physiologist, Dr. Brown-Séquard, invert the reasoning and draw chaotic and iconoclastic conclusions respecting the mode of production of hemiplegia (and other symptoms of brain disease). Starting with the generally received doctrine (never proven in a scientific way) that the pyramidal decussation is invariable and nearly total, and finding scattered in medical literature more than three hundred cases in which paralytic symptoms appeared on the same site as the lesion, he concludes that we are all wrong in considering hemiplegia to be due to destruction of or pressure upon motor tracts in the brain and basis cerebri.

Bearing in mind Flechsig's demonstration, the erroneous logic of the above is evident. Besides, these exceptional cases, so laboriously collated, should be looked at in another way. Most of them are old cases, *i.e.*, cases reported before modern cerebral anatomy was understood; many of them are related by men utterly or relatively unknown. Furthermore, any one who has worked much in the post-mortem room will appreciate how easily right is wrongly written, when left is meant, as a brain is turned over and around for examination. Consequently, we might claim the right to reject as worthless many of these three hun-

dred or more cases. Suppose, however, that they are accepted as *bona fide* examples of palsy on the same side as an encephalic lesion, and that we compare them with the thousands of cases of classical hemiplegia on the side opposite the cerebral lesion, do we obtain a proportion greater than that observed in Flechsig's series of fœtuses, one to sixty? I think not.

I have thus digressed from proper medulla diseases in order to treat the subject of pyramidal decussation fully, *i.e.*, anatomically and clinically, in one lecture. Having done this will save much repetition in treating of lesions above this point.

The pyramids whose decussation we have studied are the direct motor tracts which connect the cerebral cortex with the spinal cord. They are externally visible on the anterior (inferior) surface of the medulla, can be traced in the pons and crura, and thence into the internal capsule in the white centre of the hemispheres, to those parts of the cerebral cortex which are now considered as in some way motor. In subsequent lectures I shall describe this great motor tract more fully, and give you the reasons for believing it to be continuous from the cortex to the end of the spinal cord. The remainder of the kinesodic system of the medulla embraces longitudinal bundles of fibres which lie between the two olivary bodies (supposed to be in connection with the ganglia at the base of the cerebrum) and the various nuclei of motor nerves in the floor of the fourth ventricle. These nuclei, or groups of motor (and trophic?) cells, represent the anterior horn cells of the spinal cord, strangely thrown backward and toward the median line. They differ from the cell-groups of the anterior horns also in being more differentiated in relation to the nerves which arise from them. Reckoning from below upward, we find the nuclei of origin of the spinal accessory (11th), hypoglossal (12th), facial (7th), and abducens (6th) nerves. The last two lie on the confines of the medulla and pons. These nerves and their nuclei must, I think, be looked upon as active in three ways: 1, by their own (trophic?) neurility; 2, by reflex action set up through the adjacent and correlated sensory nerves; 3, by impulses coming from supra-bulbar

parts, great basal ganglia, cortex of the brain. This last connection is undoubtedly a crossed one, *i.e.*, the fibres or paths which transmit the motor impulse down to these nuclei cross the median line at some unknown distance above them. There is therefore another motor decussation besides the great one described above.

The æsthesodic system of the medulla oblongata occupies its lateral portions chiefly, and a part of the gray matter under the floor of the fourth ventricle, outside of the series of motor nuclei. In this gray matter we find, from below upward, the nuclei of origin of the pneumogastric (10th), glosso-pharyngeal (9th), and acoustic (8th) nerves. All through the lateral region of the medulla posterior to the olivary bodies lies the great descending root of the trigeminus (5th) nerve, which meets with groups of cells throughout this region down to the uppermost parts of the spinal cord.

These various nuclei and nerves, kinesodic and æsthesodic, make up arcs for reflex actions of the most important kind, as breathing, swallowing, and (with gray matter in the pons) the state of vascular tonus. The respiratory centre (so-called) is in the nuclei of the pneumogastrics, the vaso-motor centre lies near the median line at the junction of the medulla and pons, and there are various subordinate foci or centres for important morbid actions, as the diabetic centres, the albuminuric centre, etc. Besides, the hypoglossal nuclei and nerves preside over the movements of the chief organ of speech.

I should add that the medulla oblongata has intimate though ill understood connections with the cerebellum, by means of the restiform and olivary bodies.

As regards the localization of disease in the medulla, I shall have but little to say, for the reason that, in accordance with the terms of the course, I am bound to speak of only well-determined clinical forms whose lesions can be diagnosticated. Consequently, I will say nothing of systematic lesions in the bulbar æsthesodic system, nothing of bulbar lesions in diabetes mellitus, almost nothing of focal lesions of the organ.

a. Systematic lesions of the kinesodic tract are represented by only one typical symptom group, viz.: the so-called labio-glosso-laryngeal paralysis, or bulbar

paralysis. The lesion in this disease consists of granular degeneration of the ganglion cells of the nuclei of origin of the hypoglossus, spinal accessory, and of part of the facial nerves. The alteration is very like that present in progressive muscular atrophy (systematic lesions of the spinal cord, No. 5, Lecture III.), a molecular death, by degeneration, of ganglion cells.

The symptoms of typical labio-glosso-laryngeal paralysis are strictly motor, and consist in fibrillary contractions in and atrophic paralysis of the orbicularis oris, muscles of the tongue and throat, and some muscles of the larynx. Labial sounds are interfered with, saliva dribbles from the mouth, forcible blowing or whistling becomes impossible, the tongue plays heavily in the mouth as shown by thickness of speech and by difficult mastication, the voice becomes nasal through palatal paresis, and hoarseness, almost aphonia, is produced by palsy of the laryngeal muscles. At the same time, in extreme cases, the lower facial muscles become inert, the lower jaw hangs down, and an abundance of tenacious saliva runs from the patient's mouth; speech is quite unintelligible, and swallowing performed with extreme difficulty. At the close of life, if that be not cut short by the lodgment of food in the larynx, there are symptoms of injury to the pneumogastric nucleus, such as exceedingly rapid pulse and sudden stoppage of respiration.

In certain non-typical cases the lips, tongue and throat are affected in very various degrees; in others the pneumogastrics suffer early; and in others still the symptoms of progressive muscular atrophy, or of amyotrophic lateral sclerosis (Lecture II.) set in simultaneously or subsequently. I am disposed to believe that we may have many forms of "bulbar paralysis," and consequently would use the term as a generic one, to embrace certain varieties, only one of which is now well known.

[It will be asked why I do not speak at length of descending degeneration through the medulla. This lesion is the continuation downward of a degenerative change in the motor tract beginning in the cerebral cortex, the internal capsule, the nuclei of the corpus striatum, or in the basis cerebri.

It usually affects only one anterior pyramid, and is continued downward into the cord, in its direct and crossed pyramidal columns. No symptoms indicate this alteration in the medulla, and we infer its existence from the diagnosis of descending degeneration in the spinal cord (Section 3, Lecture II.). I do not formally treat of this systematic bulbar lesion for the reason that it has no semeiology.]

b. With respect to focal lesions. A lesion involving one lateral half of the medulla will produce, first, symptoms of injury to the kinesodic and æsthesodic systems in that half of the medulla, the symptoms being on the same side as the lesion; and second, hemiplegia of motion (and sensation, if the injury be deep enough) in the opposite side of the body. Again, a superficial focal lesion involving the anterior (inferior) face of the medulla may produce symptoms closely resembling those of bulbar paralysis of the type labio-glosso-laryngeal paralysis; and perhaps the only way of reaching a correct diagnosis is by determining the presence of the degeneration-reaction in the paralyzed facial and lingual muscles, and by demonstrating weakness or positive palsy of the extremities.

An exceedingly atypical form of bulbar paralysis is the one caused by ischæmia of the medulla, owing to arrest of circulation in the vertebral and anterior spinal arteries. The symptoms are mixed motor and sensory (deglutition and articulation impaired, breathing of the Cheyne-Stokes type, rapid pulse, tottering gait or marked general paralysis) and death rapidly ensues.

Lecture V.

SUMMARY:—LESIONS OF THE BASIS CEREBRI; PHYSIOLOGICAL ANATOMY OF THE PARTS INVOLVED. 1. LESIONS OF THE PONS VAROLII. 2. LESIONS OF THE CRURA CEREBRI. 3. LESIONS OF THE BASAL PARTS FORWARD OF THE CRURA; HEMIOPIA AND NEURO-RETINITIS.

GENTLEMEN:—Leaving the region of the medulla, we are brought to those numerous and important parts

which constitute the encephalon. I purpose considering in some detail, in this and subsequent lectures, the localization of disease in the chief subdivisions of this mass, but in this lecture I can only enunciate some general pathological propositions relative to the encephalon as a whole, and study the lesions of one of its parts.

For our purpose I make the following subdivision of the encephalon—a semi-physiological classification:

1. The basis cerebri, including all the parts which lie upon the base of the skull, but more especially the pons Varolii, crura cerebri, their attached nerves, and the optic and olfactory apparatuses.

2. The great basal ganglia; *i.e.*, the thalamus opticus, nucleus caudatus, nucleus lenticularis, and the corpus quadrigeminum.

3. The white substance of the hemispheres, especially the internal capsule.

4. The cortex cerebri.

5. The cerebellum.

The general pathological propositions relative to these parts are as follows:

1. Lesions of the basis cerebri, especially if involving the pons and crura, give rise to the following symptoms: paralysis (often of crossed variety), anæsthesia in the face and limbs, impairment of equilibrium, changes within the eyes; no psychical symptoms.

2. Lesions of the great basal ganglia probably produce no symptoms unless by encroaching upon the internal capsule which passes near them. An exception may be the nucleus caudatus.

3. Lesions of the white centre of the hemispheres produce no symptoms when they do not involve the parts composing the internal capsule; if the anterior portion of this capsule be injured, we observe paralysis, if its posterior part, anæsthesia.

4. Lesions of the cortex cerebri produce, when located anteriorly, psychical symptoms; when located in the median regions, paralysis of an imperfect kind, and when situated posteriorly, no symptoms at all (sensory symptoms in animals).

5. Lesions of the cerebellum produce no symptoms

except by involving adjacent parts containing important motor and sensory tracts; thus giving rise to irregular paralyses, changes in the optic apparatus, symptoms of irritation of the vagus nerve, etc.

6. Lesions in one lateral half of any part of the encephalon produce motor and sensory symptoms in the side of the body opposite to the lesion. When the lesion is in one-half of the basis cerebri some symptoms (direct symptoms) are found in the side of the face and head corresponding to the lesion, others in the opposite half of the body (crossed paralysis).

7. Lesions in the median line cause symptoms to appear in both sides of the body.

8. Any intracranial lesion which acts in such a way as to increase the intracranial pressure may produce (in addition to other symptoms) the condition known as choked disk, or neuro-retinitis.

With these preliminary general statements, I pass to the study of the first of the subdivisions—the basis cerebri:

PHYSIOLOGICAL ANATOMY.

To be very logical, the medulla oblongata, pons, and crura should be grouped together under this denomination, but for clinical purposes I leave the medulla with the spinal cord, and add to the basis cerebri the parts which lie in front of the crura, viz., the optic tracts and nerves, and the olfactory apparatus.

As heretofore, I assume that you are familiar with the descriptive anatomy of the parts; and what I wish you particularly to understand is the arrangement of sensory and motor tracts, of nerves, and of ganglia in the basis cerebri.

The projecting mass we call the pons is largely made up of numerous transverse nerve-fibres which connect it with the two halves of the cerebellum, and which are not, under our present knowledge, of special physiological or pathological importance. In the anterior (inferior) half of the pons, lying under those transverse fibres, and partly separated into bundles by them, we find the great motor or pedimcular tract, the continuation upward of the anterior pyramids and of the central motor fasciculi of the medulla. This tract

is easily seen with the naked eye, broken up into a number of smaller fasciculi by transverse fibres, and it can be traced upward a long distance. In the posterior (superior) region of the pons, under the floor of the fourth ventricle, near the median line, there is a lengthened mass of motor gray matter, whence arise the original fibres of the sixth, seventh, and motor root of the trigeminus nerves. Laterally, in the posterior part of the pons, are sensory tracts, some directly continuous with the brain above, others more particularly in relation with the sensory root of the trigeminus, which radiates to an immense extent up and down in the basis cerebri and below it.

In the crura cerebri we find substantially the same parts not covered over by transverse fibres. The anterior portion of the crura consists of the great motor or peduncular tract just issued above from the white matter and basal ganglia of the cerebrum. Posteriorly are sensory tracts and masses of gray matter in intimate relation with the thalamus opticus and corpus quadrigeminum. The final (upper) extremity of the elongated mass of gray matter giving rise to motor nerves is found here, posteriorly, just beneath the aqueduct of Sylvius, in the shape of the common nucleus of the third and fourth nerves. Let us finally bear in mind that the sixth, seventh, and fifth nerves are intimately connected with the pons Varolii; the third and fourth nerves, and the optic tracts are associated with the crura.

The posterior portions of the crura and pons constitute the greater part of what Meynert designates the *tegmentum cruris cerebri*, while their anterior parts contribute to form his *basis cruris cerebri*.

All these parts lie in the middle fossæ of the cranium, the chiasm of the optic nerve occupying their forward extremity. In the anterior fossæ we find only the olfactory commissures and ganglia, together with the under surface of the frontal lobes of the cerebrum.

As regards the physiology of these parts, the mesencephalon of some authors, we may sum it up as follows:

The anterior part of the crura, pons (apart from the superficial transverse fibres), and medulla oblongata, contains the chief motor tract connecting the superior

centres with the spinal cord. This motor tract is made up of nerve fibres, which convey excitations chiefly in a centrifugal direction; many of them, derived from the motor regions of the cortex, constitute the anterior portion of the internal capsule, and then enter the crura, traverse the pons, go to make up the anterior pyramids of the medulla, partially and irregularly decussate at the pyramidal decussation, and finally are found in the postero-lateral and anterior columns of the spinal cord. This most important bundle of nerves may be designated the *direct cerebral motor tract*. Another portion of the anterior region of the mesencephalon is likewise motor in function, and is made up of bundles of fibres derived from the nucleus caudatus and nucleus lenticularis. These bundles can be traced downward into the middle regions of the medulla, but their connection with the spinal cord is yet uncertain. The posterior region of the pons and crura, composed largely of gray matter, is partly sensory and partly motor. It is motor only in so far as it includes the nuclei of origin of the upper cranial nerves, the seventh, sixth, motor root of fifth, the fourth and third. You will remember that the nuclei of these nerves are all to be found near the median line, underneath the floor of the fourth ventricle or its continuation, the aqueduct of Sylvius. The sensory parts of the mesencephalon embrace the regions lying external to these nerves, giving origin to the eighth and fifth pairs of nerves; the latter presenting an enormous expansion in its origin; its upper roots extending as high as the region of the corpus quadrigeminum, the lowest probably as far down as the upper part of the spinal cord.

The posterior regions of the mesencephalon, the *tegmentum cruris*, are the seat of the reflex actions of the most important character, and they probably serve also for the elaboration, if not perception of sensory impressions from the periphery. At the upper part of the medulla and the lower part of the pons in this region is a mass of gray matter which controls the vaso-motor phenomena throughout the body. It is highly probable that some severe convulsive manifestations, such as epileptic and tetanic seiz-

ures. are due to morbid processes in the posterior part of the pons and crura.

Finally, with respect to the physiology of the optic apparatus lying at the base of the brain, I will only say that I am disposed to accept the doctrine of semi-decussation of the optic tracts in the chiasm, and shall use this hypothesis in explanation of symptoms.

SYMPTOMS OF LESIONS OF THE BASIS CEREBRI.

Before taking up systematically the study of the semeiology of basal lesions, allow me to fully discuss one of the most frequent symptoms of all these lesions, viz., crossed paralysis. I wish to study crossed paralysis in general, previous to speaking of it under each heading of the remainder of this lecture, partly to impress you with its importance, and partly to avoid future digressions and repetitions. By crossed paralysis (*paralysie alterne* of the French) is meant a form of paralysis in which the symptoms immediately caused by a basal lesion are on one side of the face or head and on the same side as the lesion, while the bodily symptoms are on the opposite side of the median line, viz., on the side opposite the lesion. As thus enunciated, in principle, it is at once apparent that the phenomena of crossed paralysis may, according to the seat of a lesion at the base of the brain, involve, on the one hand, any one of the cranial nerves, and on the other the limbs of the opposite side. This conception is verified by clinical and post-mortem experience, cases of crossed paralysis of every possible variety being on record. The most striking and best known, however, are those presenting what I may be allowed to term the third nerve and body type, the trigeminus and body type, and the seventh nerve and body type. It is to the late Prof. Romberg, of Berlin, that we owe the exact definition and conception of the principle of crossed paralysis, while Prof. Gubler, of Paris, first made a thorough study of the seventh nerve and body type.

1. LESIONS OF THE PONS VAROLII.

Diffused lesions of the pons produce, when fully developed, partial anæsthesia and paresis on both

sides of the face, generalized paralysis below the neck, with or without anæsthesia, the latter symptom appearing only if the deeper, posterior, portions of the organ are involved. We also observe inability to maintain equilibrium, without ataxia, sometimes convulsions, or contracted pupils, or neuro-retinitis. If the lesion advance forward beyond the pons, new symptoms, such as will be described further on, are superadded.

Localized lesions of the pons may occupy on either side of the median line one of four locations.

a. In the anterior portions of the pons forward of an imaginary transverse line passing through the origin of the trigemini. As shown by Gubler, this lesion does not produce crossed paralysis of the seventh nerve and body type, but both the face and body are paralyzed on the same side, *i.e.*, on the side opposite the lesion, just as in cerebral lesions strictly speaking. A point for differential diagnosis is that when the cerebrum is injured, there is almost invariably conjugate deviation of the head and eyes toward the affected hemisphere; in pons lesions nothing of the kind occurs.

b. A lesion placed so as to injure the anterior (inferior) region of the pons on one side, back of the imaginary line above mentioned, will cause typical crossed paralysis, *i.e.*, the facial nerve will be paralyzed on the same side as the lesion, and the extremities on the opposite side. For example, if the right face and the left arm and leg be paralyzed in a patient, we recognize crossed paralysis of the seventh nerve and body type, and may diagnosticate with positiveness a lesion placed as above described. There is, perhaps, no more positive example of constant relation of lesion to symptoms in the whole of nervous pathology.

In *a* and *b*, if the trunk of the trigeminus be involved in the disease, the face will be more or less anæsthetic and neuralgic on the same side as the facial palsy and the lesion. From a study of cases of these two kinds, Gubler drew the anatomical conclusion that the paths which connect the cerebrum with the nuclei of the seventh nerve decussate at about the middle of the pons, *i.e.*, some distance above the nuclei

of these nerves. A useful diagram for studying the principle of crossed paralysis, and of this type in particular, you will find in Dr. Hammond's Treatise on Diseases of the Nervous System, ed. 1876, p. 99.

c. Lesions occupying the posterior region of the pons above its middle. Besides paralysis of the face and body on the side opposite the lesion, we are likely to have anæsthesia of the paralyzed parts, even amounting to hemi-anæsthesia (without involvement of the special senses.) Other symptoms often produced are epileptic convulsions, impairment of sight from neuro-retinitis, and various forms of paralysis of ocular muscles.

d. Lesions in the posterior part of the pons, below the imaginary transverse line through its equator, may likewise, if extensive, without crossing the median line, cause hemi anæsthesia of common sensory nerves, but will also produce crossed palsy, of face on same side as the lesion, of the body on the opposite side. As in *c*, we may have neuro-retinitis, epileptic seizures, with, besides, palsy of the sixth nerve, and bulbar symptoms if the lesion involve the medulla.

e. A lesion situated very laterally in the pons, or so placed as to irritate the lateral peduncles of the cerebellum, will (as shown by a number of cases) give rise to rotatory movements of the patient around the long axis of his body toward the side of the lesion. Of course this symptom appears with others, which are more or less in accord with the above symptom-groups.

2. LESIONS OF THE CRURA CEREBRI.

a. Lesions of the crus proper on one side of the median line. The symptoms of such a lesion are exceedingly definite, and might even be designated pathognomonic. The third nerve, its trunk or origin, is involved in the disease or compressed, as well as the great motor tract which, lower down, is to decussate. Consequently we observe a crossed paralysis of the third nerve and body type; *i. e.*, if the right motor oculi and the left extremities be paralyzed in a patient, we may feel sure that he has a lesion under or in the right crus cerebri. Prof. Rosenthal, of Vienna, states

that in such cases the electro-muscular contractility (to faradism) is reduced in the paralyzed limbs, contrary to what obtains in hemispheric lesions. You will remember that the optic tract curves around the crus on either side, and it at times happens that symptoms characteristic of injury to one optic tract present themselves. These symptoms will be considered later.

b. Lesions deeply placed in the crus, on one side of the median line. In addition to the above described crossed paralysis, we shall probably observe hemi-anæsthesia of common sensation only on the side opposite the lesion. There may be convulsions, choreiform spasm, hemiopia, or neuro-retinitis.

c. Lesions placed in the median line, or involving both crura more or less. The symptoms will be, in case of lesion situated anteriorly (inferiorly), wholly motor, viz.: paralysis of both third nerves, and of both sides of the body below the neck. In case of lesion involving the tegmentum cruris, marked disorders of sensibility, neuro-retinitis, and convulsions will occur.

Extension of the disease backward to the pons will be characterized by symptoms (detailed in first paragraph) referable to the trigeminus and seventh nerves.

It has been stated that the bladder is paralyzed in severe lesions of the crura, but this lacks confirmation.

It might not be ill to add that patients having lesions in the locations defined above, do not present, strictly speaking, cerebral symptoms. They preserve their intellect, are not aphasic, and the special senses (except the sense of sight) are not involved.

3. Lesions situated at the base of the brain anteriorly to the crura.

These are rare, but give rise, when considerable, to noteworthy symptoms.

a. Lesions involving one of the optic tracts posterior to the chiasm. A small lesion in this location will produce hemiopia, a symptom of such importance and

scientific interest as to require special mention. If the lesion extend, it may involve the chiasm of the optic nerves, or the tract of the opposite side, or the crura cerebri, thus either destroying the special symptom, hemiopia, or adding to it the signs of crossed paralysis of third nerve and body type.

By hemiopia is meant a condition of the organs of vision such that only one-half (vertically divided) of objects are seen by the patient. In other words, the fields of vision of the two eyes are darkened in one of their halves. Usually the vertical line of division between the lighted and darkened half-fields is exactly in the centre of vision. The hemiopia affects the two eyes always, and is distributed differently in each eye under the determining influence of the exact location of lesions about the optic tracts and chiasm. The immediate cause of hemiopia is interruption in the centripetal conducting power of certain bundles of nerve-fibres in the optic nerves and tracts; hence it has been found necessary to frame a theory of the course of fibres in the optic apparatus. The hypothesis of Wollaston, that of semi-decussation, is the one which is generally adopted to-day, and I append a diagram illustrating it, and the positions of lesions producing the varieties of hemiopia. A satisfactory anatomical demonstration of the truth of the theory of semi-decussation has not yet been given, and some few facts and authorities are opposed to it, but I think that I can assure you that in the present state of science it is the one which best serves the purposes of the physiologist and the clinical observer.

According to this hypothesis nerve-fibres derived from the deep origins of one (the left) optic tract, combine to form that bundle and extend undivided as far as the chiasm. As shown in Fig. 6 at the chiasm some of the fibres of the tract (the external group possibly) pass directly into the external portions of the left optic nerve, and are distributed to the external (temporal) half of the left retina. The remaining nerve-fibres of the left optic tract cross the median line in the middle of the chiasm, decussating with the similar fibres derived from the right tract, then enter the right optic nerve, and are distributed to the inner

(nasal) half of the right eye. Thus it is seen that the nasal half of the right eye and the temporal half of the left eye are anatomically and physiologically homologous.

The same explanation applies to the course of fibres which form the right optic tract. Besides these main bundles of fibres, a few filaments are supposed to connect the two retinæ, passing through the optic nerves and the anterior border of the chiasm without decussation, while others connect the two sets of tubercula quadrigemina by way of the optic tracts and the posterior border of the chiasm. These commissural fibres may, however, be left out of consideration in the study of hemiopia.

If now we bear in mind this distribution of fibres in the optic apparatus we can by the aid of Fig. 6 demonstrate the mechanism of each of the four forms of hemiopia, viz.: right and left lateral hemiopia (shaded parts A and B), bi-temporal and nasal hemiopia.

Lateral hemiopia, *i.e.*, that form of hemiopia in which the homologous parts of the two eyes (temporal half of the left retina and the nasal half of the right) lose their function, is invariably produced by a lesion destroying one optic tract—the left in the diagram. *Vice versa*, destruction of any part of the right optic tract would cause blindness in the temporal half of the right retina and in the nasal half of the left. Inasmuch as all visual rays cross in the eye owing to refraction in the lens, the hemiopia, *i.e.*, the obscuration of the half-fields of vision is on the opposite side from the blind half-retinæ. For example, in the first instance described above, and illustrated in the diagram, the hemiopia is of the form designated right lateral (or homonymous) hemiopia, and is produced by a lesion involving the left tractus opticus.

Binocular temporal hemiopia can be caused only by a lesion placed (as 1 in Fig. 6) in front of the chiasm, cutting off the fibres which supply the two nasal fields.

Binocular nasal hemiopia is produced by a double lesion compressing or destroying the outer parts of the chiasm (2 in Fig. 6), which are imbedded fibres supplying the temporal halves of both retinæ. Prof. Knapp, of New York, has placed a remarkable case

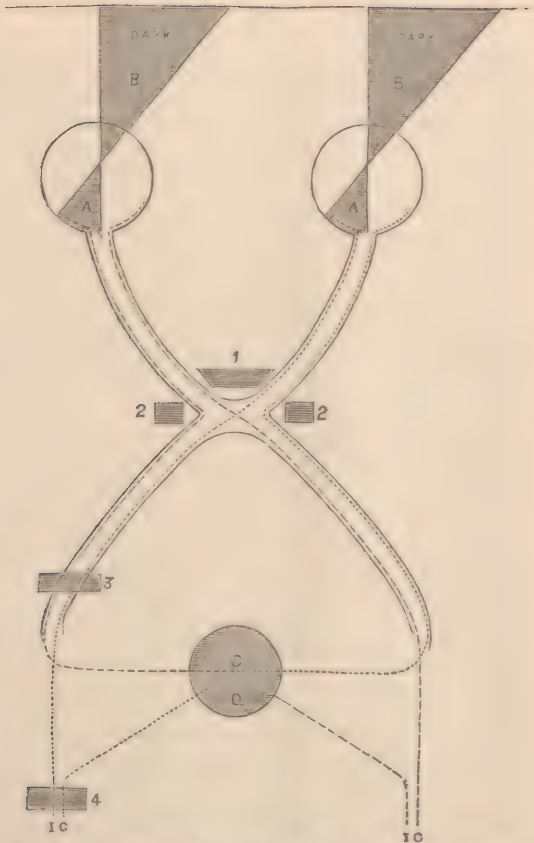

FIG. 6.—Diagram explicative of hemiopia. The shaded intra and extra ocular parts, A and B, indicate the obscuration in right lateral (or homonymous) hemiopia, as caused by lesion 3, so placed as to destroy one optic tract. In that tract are two sets of nerve fibres, one represented by a dotted line supplying the nasal half of right retina, the other fibres by a broken line supplying the outer or temporal half of the left eye. As visual lines cross in the eye the obscuration of the half-fields is the opposite. Lesion No. 1, anterior to chiasm, produces blindness of inner half of each retina, and consequently bi temporal hemiopia. Lesions No. 2, pressing upon the sides of the chiasm, injure fibres supplying the temporal half of each retina, and cause bi-nasal hemiopia. C. Q. corpus quadrigeminum, in which Prof. Charcot believes a second partial decussation takes place. 1 C. Internal capsule containing, on Charcot's hypothesis, all the fibres coming from the eye of the opposite side. 4. Lesion of internal capsule injuring all the fibres connected with the right retina, and causing amblyopia of the right eye.

of this kind on record, in which the lesion consisted of thickened and enlarged internal carotid arteries. I need hardly add that lesions involving one optic nerve in front of the chiasm cannot give rise to hemiopia; they produce monocular loss of vision.

It should not be forgotten that lesions lying in front of the crura and behind the chiasm may press upon the motor nerves of the eye as they traverse the middle fossæ of the cranium on their way to the sphenoidal fissure, thus producing a variety of paralytic **symptoms about one or both eyes.**

b. Lesions in the anterior cranial fossa, anterior to the chiasma. When unilateral, such a lesion involves the olfactory tract and ganglion, thereby producing anosmia, or loss of smell, on the same side as the lesion, **with or without subjective odors.**

If the lesion be large, it may act upon the nucleus caudatus or other motor parts of the encephalon, and cause common hemiplegia (face and limbs) on the opposite side. Thus we may have a last form of crossed paralysis, of the olfactory nerve and body type. With a lesion involving both sides of the median line, complete double anosmia, with or without generalized paresis of the extremities, would be met with.

There is one more symptom common to all lesions of the basis cerebri, but produced also at times by any intracranial disease which causes pressure. I mean neuro-retinitis or choked disks. This is always (?) bilateral, though it may be more marked in one eye, and is esteemed one of the most important signs of gross encephalic disease, especially of tumors. In neuro-retinitis, ophthalmoscopic examination shows that the optic nerves are swollen, and they may project considerably (measurably) above the level of the surrounding retina; the margin of the disk is obscured or wholly lost, and no line of demarcation can be made out between the nerve and the retina. The blood-vessels present striking anomalies, the arteries being relatively small, the veins positively enlarged and tortuous; there are often small hæmorrhages in the retina, round about the disk. Strange to say, very good sight may coexist with this lesion. This condition of choked disks may last a number of

weeks (much longer in cases of tumor of the brain), and then subside, giving place to the appearances of atrophy of the optic nerves, viz., an unnatural whiteness or bluish whiteness of the disk, smallness of the retinal vessels, and unusual sharpness of the outline of the disk, with impaired vision.

Lecture VI.

SUMMARY:—LESIONS OF THE GREAT GANGLIA AT THE BASE OF THE BRAIN, AND OF THE WHITE SUBSTANCE OF THE HEMISPHERES.—SKETCH OF THE PHYSIOLOGICAL ANATOMY OF THESE PARTS: LESIONS OF THE GREAT BASAL GANGLIA; OF THE INTERNAL CAPSULE, AND OF THE REMAINDER OF THE WHITE SUBSTANCE.—LESIONS OF THE CEREBELLUM.

GENTLEMEN:—The great ganglia at the base of the brain are from before backward, the nucleus caudatus and the nucleus lenticularis of the corpus striatum (the intra- and extra-ventricular portions of the corpus striatum, according to English and American books), the thalamus opticus, and the corpus quadrigeminum (or tubercula quadrigemina). These masses of gray matter are of course double, *i. e.*, symmetrically arranged on either side of the median line. Their relations to adjacent portions of the brain are of great importance to us, and worthy of a somewhat detailed study.

In very general terms, it may be said that all of these bodies have at least a duplex connection, one superior with the cortex of the brain, the other inferior with the various parts which make up the mesencephalon. More particularly, the nucleus caudatus (intra-ventricular nucleus of the corpus striatum) sends bundles of fibres downward into the crura, and the same is true of the externally-placed nucleus lenticularis. So we say that both parts of the corpus striatum are intimately connected with the basis cruris cerebri.

On the other hand, the thalamus and corpus quadrigeminum are intimately united with the nucleus of the tegmentum, and send bundles of fibres into and

through it, in the posterior or sensory system of the mesencephalon. Physiologically, we may look upon the basis cruris and its superadded ganglia as motor in function, and upon the tegmentum cruris as sensory and as the seat of performance of important and complex reflexes.

As regards the other connections of these bodies, they are probably connected with their homologues across the median line, and superiorly with various parts of the cortex. There would seem to be, judging from a series of cases of cerebral atrophy, a bundle of nerve-fibres connecting the nucleus caudatus of one side with the opposite half of the cerebellum by way of the processus cerebelli ad cerebrum. At any rate, great atrophy of the right hemisphere (for example) and corpus striatum is usually accompanied by atrophy of the left hemisphere of the cerebellum. I need hardly remind you of the intimate union between the optic tracts and the corpus quadrigeminum and the external portion of the thalamus.

The white centre of the hemispheres is made up of many separable fasciculi, the physiology of many of which is as yet obscure. In the first place, we can demonstrate in it commissural bundles running in various directions: transversely, connecting various portions of the cortex of both hemispheres by way of the corpus callosum and the so-called commissures; others extend longitudinally in one hemisphere, connecting the convolutions of one lobe with those of another; yet others simply bind together adjacent convolutions. Secondly, there are heavy masses of fibres extending from the basal ganglia to the convolutions of the hemispheres, constituting a great part of what is known as the corona radiata. Thirdly, and most important in pathology to-day, is a bundle of white substance in each hemisphere, which directly unites the cortex of the brain with the crura, pons, and spinal cord — the so-called internal capsule. This fasciculus appears (upon anatomical and physiological evidence) to be the continuation of the sensory and motor tracts which we have studied in the basis cerebri. The extension of the sensory tract upward from the crura into the internal capsule is not so

clear as is the continuity of the motor tract anteriorly. This, denominated by some the peduncular tract, and designated in Lect. V. as the *direct cerebral motor tract*, can be traced by dissections, by physiological experiments, by embryology, and lastly by the help of pathological processes (descending degeneration) from certain convolutions of the cortex

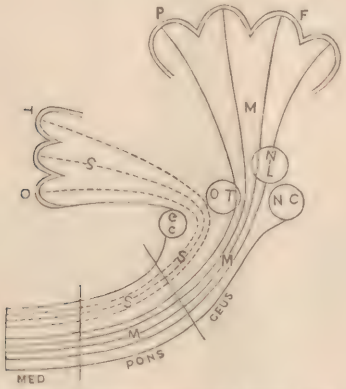

Fig. 7.—Diagram of course of sensory and motor tracts in the mesencephalon and hemispheres. S, sensory tract in posterior region of mesencephalon, extending to O and T, occipital and temporal lobes of hemispheres; M, motor tract in basis cruris, extending to P and F, parietal and (part of) frontal lobes of hemispheres; C C, corpus quadrigeminum; O T, optic thalamus; N L, nucleus lenticularis; N C, nucleus caudatus.

cerebri into the anterior half or two-thirds of the internal capsule, into the crus cerebri, the pons, the medulla, and (in accordance with the law of decussation—*vide* Lect. IV.), into both halves of the spinal cord. The composition and extension of the internal capsule is rudely represented in diagram by Fig. 7. Its exact position in the hemisphere and its relations to other parts of the encephalon is well shown in Fig. 8, modified from a cut in Prof. Charcot's Lectures on Localization in Cerebral Diseases. This represents a transverse vertical section of the hemispheres made

through the middle of the optic thalamus. The internal capsule is seen to lie between the external border of the thalamus and the nucleus lenticularis of the corpus striatum. In this location the bundle is much compressed, and seems to have little or no connection

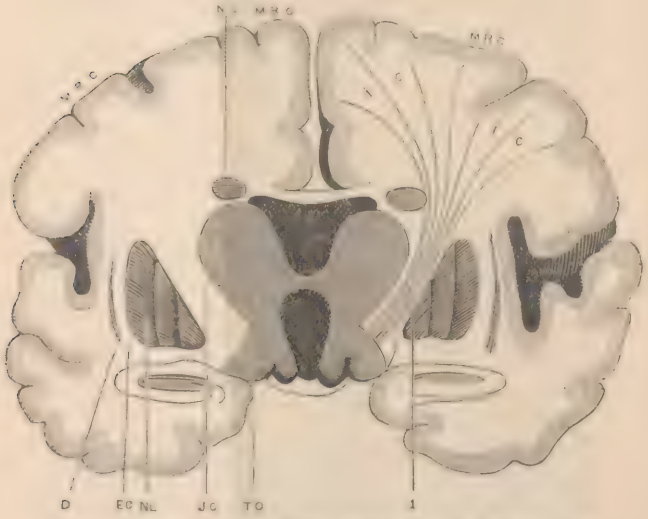

FIG. 8.—Modified from Charcot's diagram, to show position, relation, and distribution of the internal capsule as seen in a vertical transverse section of the brain on a level with the greatest development of T O, thalamus opticus. I C, location of internal capsule; N L, nucleus lenticularis; E C, external capsule: D, claustrum; N C, nucleus caudatus; M B C, motor regions of cortex cerebri; 1, fibres representing the radiation of the internal capsule ventrally to the motor region of cortex.

with the gray bodies just named; it expands in every direction after passing them, and its anterior fibres are ultimately distributed to the middle regions of the cortex cerebri—motor district—embracing the third frontal, the posterior extremities of the first and second frontal, the ascending frontal convolutions, also the ascending parietal and first parietal convolu-

tions; also, lastly, to the continuation upon the inner surface of the hemisphere of the ascending frontal and parietal convolutions which make up the paracentral lobule. The posterior third of the internal capsule is very probably distributed to the occipital, temporal and second parietal convolutions—the supposed sensory gyri.

Besides common sensory tracts, the posterior part of the internal capsule contains fibres which are directly or indirectly connected with the special sense organs, viz., the optic, olfactory, gustatory, and acoustic. This is shown by the fact that total hemi-anæsthesia in man results from lesions involving the internal capsule: from experiments upon animals, whereby certain occipital or temporal convolutions being removed, blindness or deafness is caused on the opposite side.

The physiology of the great ganglia, commissural fibres, and internal capsule is far from satisfactorily marked out. Many glaring contradictions are apparently proved by the experiments of different observers. It would appear well settled that the corpus quadrigeminum and the corpora geniculata are a part of the optic apparatus; that the nucleus caudatus and the nucleus lenticularis have motor functions of some sort. The greatest uncertainty exists, I think, concerning the attributes of the thalami optici.

On the other hand, we now know pretty positively that most of the sensory paths for the body pass in the posterior part of the internal capsule, laceration of this part in animals being invariably followed by hemi-anæsthesia on the opposite side. That the thalamus has anything to do with the perception of sensations is rendered doubtful by the occurrence of cases in which lesions, strictly limited (*i. e.*, not pressing on adjacent parts) to the thalamus have produced no special symptoms. The same objection (pathological) can be urged against the view that the nucleus lenticularis has important motor functions. That the anterior part (two-thirds?) of the capsule consists of motor fibres is likewise quite well established by experiments upon animals and by the study of patho-

logical cases.* What the functions of the various commissural fibres may be, is, at the present time, only a matter of speculation more or less logically constructed.

1. LESIONS OF THE BASAL GANGLIA.

The generally received statements that lesions of these bodies produce definite symptoms—lesions of the nucleus lenticularis and nucleus caudatus paralysis, and lesions of the thalamus opticus anæsthesia—are, I think, very questionable. From the evidence now before us it seems doubtful if lesions of the nucleus lenticularis, and of the thalamus, produce any symptoms except by exerting pressure upon the internal capsule lying near by. At any rate, cases of destruction of large parts of these bodies without symptoms are on record. As regards the nucleus caudatus, it is possible that its destruction is followed by hemiplegia and secondary degeneration; but on the other hand, it must be admitted that almost all lesions of this body are so placed, and of such a nature, as to cause pressure upon the motor portion of the internal capsule. This is especially true of hemorrhage, as shown by Prof. Charcot in his recent lectures on the brain.

2. LESIONS OF THE INTERNAL CAPSULE.

These, on the other hand, produce, as shown by the recent study of pathological evidence, and by experiments upon animals, constant symptoms. If the anterior half or two-thirds of the capsule be injured, we have hemiplegia on the opposite side of the body;

* Since these lectures were delivered, a brilliant and final proof of the motor functions of the anterior part of the internal capsule, and of the continuity of the motor convolutions and the direct cerebral motor tract has been advanced by the French experimenters, Franck and Pitrès (*Le Progrès Médical*, Jan. 19, 1878). These physiologists found that by faradizing those parts of the white centre of the hemisphere which lie underneath the so-called cortical motor centres, they were able to produce definite movements in parts of the body on the opposite side. This tract of white matter, constituting the anterior portion of the internal capsule, contains physiologically distinct fasciculi which are connected on the one hand with the motor districts of the cortex, and on the other hand with peripheral parts of the body across the median line.

more or less perfect hemiplegia according to the exact seat and size of the lesion. It will be readily understood that lesions (especially hæmorrhage and tumors) of parts adjacent to the capsule, such as the lenticular and caudate nuclei, the white centre of the frontal lobe, etc., may by pressure bring about a similar result. Besides paralysis, descending secondary degeneration is an inevitable result of lesions of the internal capsule. This lesion can be traced downward through the whole length of the direct cerebral motor tract so frequently referred to before, to the lower end of the spinal cord. Lesions of the nucleus caudatus are also said to produce the same results, but recent researches (Flechsig) throw doubt on this.

If the posterior part of the internal capsule be injured directly, or indirectly by the pressure of lesions in adjoining regions, there is produced anæsthesia, more or less complete, on the opposite side, usually with only slight paralysis. If the lesion be considerable the anæsthesia is absolute; *i. e.*, the special senses and common sensory nerves lose their function, or more properly speaking, impressions coming through these cannot reach the perceptive centres. Hemiopia is never (?) thus produced, and Charcot explains this by supposing a second semi-decussation to take place in some part between the chiasm and the internal capsule; probably in the corpus quadrigeminum. A reference to Fig. 6, Lect. V., will, I trust, make this hypothesis plain.

Yet a third symptom results from lesions of the internal capsule, viz., choreiform movements following hemiplegia and hemi-anæsthesia. These movements vary in degree and type from true athetosis to ataxia, from chorea to tremor, and constitute an interesting symptom group, well worthy of further study. In my own cases of post-hemiplegic chorea, hemi-anæsthesia of slight degree was present, and in one case lateral hemiopia.

Extensive lesions of the central parts of the hemispheres may produce, besides the specific signs named above, a number of other symptoms. Thus, suddenly produced lesions (hæmorrhage, softening) will nearly always cause conjugate deviation of the eyes

and head. The patient, though insensible, turns his eyes and head constantly to one side, toward the injured hemisphere, and away from the paralyzed side. In spite of a recent attempt to impeach the value of this symptom of hemispheric injury, I am disposed to attach value to it. Great increase in the bodily temperature also follows large injuries.

Other lesions, such as cause pressure, and slowly grow to a great size (tumors), cause, in addition to the specific signs dependent upon their exact location, the change within the eye which we call neuro-retinitis, (*vide* Lect. V.). They will also usually produce convulsions.

As regards the remainder of the white substance, such as the central regions of the frontal, occipital, and temporal lobes, modern critical study of recorded cases would seem to indicate that lesions involving these parts in such a way as not to press upon the internal capsule and nucleus caudatus, do not give rise to any symptoms; *e. g.*, an immense abscess may occupy the temporal and occipital lobes, or the anterior part of the frontal lobe, without causing paralysis or anæsthesia. Anatomy and experimentation, however, seem to indicate that lesions of the occipital and temporal lobes should give rise to sensory symptoms; and a more careful study of cases of disease in these parts is just now a desideratum.

We do not know any more relative to lesion of the great commissural bundles which unite the two hemispheres and different parts of one hemisphere. The cases of congenital absence of the corpus callosum on record do not teach anything definite.

3. LESIONS OF THE CEREBELLUM.

I add a few words relative to another *terra incognita* in the brain, the cerebellum. Its situation is known to all of you, but there are a few points in its anatomy to which I would specially invite your attention. In the first place, this great mass of nervous matter is closely bound down by a strong fibrous covering (a bony septum in some animals), the tentorium cerebelli. This fold of dura mater probably serves important

purposes in health, but in case of disease in the cerebellum it causes pressure-effects to be transmitted chiefly forward and downward. This is important to bear in mind when studying the effects of cerebellar lesions. Second, the cerebellum is remarkable for its numerous connections with other parts of the nervous system. Fibres connect each of its hemispheres with the nucleus caudatus and cerebral hemisphere of the opposite side, by means of the crura cerebelli ad cerebrum. Other fibres, forming heavy bundles, make up the crura ad pontem or lateral peduncles of the organ; extending deeply into the white and gray substances of the mesencephalon. It is probable that each half of the cerebellum is thus connected with the opposite half of the pons; possibly some fibres are strictly commissural, *i.e.*, unite the two hemispheres of the organ after passing over the pons. Lastly, the cerebellum is connected with the medulla and spinal cord. It forms, by means of the crura cerebelli ad medullam, close connections with the olivary bodies, and with the external portion of the lateral columns (near the extremity of the posterior horns) in the spinal cord. It has been claimed that some cranial nerves (third, fourth, and acoustic) have been traced into the cerebellum, but the evidence on this point is unsatisfactory. Third, the cerebellum overlies highly important organs, and this proximity serves to explain much of the semeiology of its lesions. In front of it is the corpus quadrigeminum and the tegmentum cruris, with its contained vaso-motor (and convulsive?) centre; beneath it the medulla oblongata, with its floor and such vital nerves as the pneumogastric and the spinal accessory.

The physiology of the cerebellum is at the present day quite unknown. That it serves for purposes of co-ordination in a direct and positive manner is disproved by experimentation and pathology; that it is a centre for the movements of the eyeballs (Ferrier) is equally doubtful; and so is the view that it is the seat of psychical attributes of an emotional character. Mitchell's hypothesis, that it is a store house of nerve force for use in emergencies, is plausible but unproven. The mechanism and purpose of the cerebellar connections is likewise not understood.

As regards the diagnosis of lesions of the cerebellum, I must admit that in the very numerous symptoms produced by them I do not know of one that is characteristic. In other words, lesions strictly limited to the substance of the cerebellum produce no definite symptoms: and on the other hand, the symptoms which we observe in cerebellar diseases are the result of pressure-effects upon adjacent parts. Thus, the affections of sight so common in cerebellar lesions are caused by pressure upon the corpus quadrigeminum or the corpora geniculata, and also upon the origins of the third, fourth and sixth nerves. The nausea, vomiting, and sudden death may be explained by irritation and paralysis of the nuclei of the pneumogastric nerves in the floor of the fourth ventricle; convulsions, by pressure upon the tegmentum cruris; the imperfect hemiplegia or general paralysis, by a similar action upon the motor regions of the mesencephalon. The diagnosis (assisted by predominance of pain in the occipital region) must be made chiefly by exclusion. A symptom of great importance when present is titubation. This has been termed cerebellar ataxia, but as a descriptive term titubation is better. The patient walks with his feet separated, his body bent a little forward and swaying, his hands and arms in use to preserve his equilibrium. There is no true ataxic jerking, no want of harmony between antagonistic groups of muscles, no choreic movements, no tremor.*

* After the delivery of this lecture Prof. H. Nothnagel of Jena, published (in *Ber. med. Gesellsch. Wochenschrift*, 1878, No. 15) the results of his analysis of more than two hundred and fifty cases of cerebellar disease. His conclusions, I am happy to say, are substantially equivalent to what has been said above. However, Prof. N. is disposed to admit one symptom—cerebellar ataxia—as characteristic of injury to the cerebellum, or more properly, to one of its smaller parts, the superior vermiform process. N. says that by cerebellar ataxia we are to understand a perversion of equilibrium closely resembling that observed in alcoholic intoxication; the patient titubates, stands with feet wide apart; if he be barefooted the toes are seen in active motion; and in walking the body sways a good deal, the foot is brought down with ball or with heel first irregularly; closing the eyes sometimes makes standing and walking worse, sometimes not. In the recumbent position there is no ataxia. In the large majority of cases the upper extremities remain free from inco ordination.

Lecture VII.

SUMMARY:—ANATOMY AND LESIONS OF THE CORTEX OF THE BRAIN.—THE CHIEF CORTICAL MOTOR CENTRES, AND BROCA'S SPEECH-CENTRE.—LOCALIZED LESIONS OF THE CORTEX CEREBRI; DIFFUSED LESIONS OF THE SAME.

Gentlemen:—The cerebral cortex is an immense spread-out ganglion, whose functions are not yet fully or exactly known. Like all ganglionic masses, it is composed of ganglion-cells, nerve-fibres, blood-vessels, and neuroglia. Its ganglion-cells are generally pyramidal in shape, the apex of the pyramids being turned outward or peripherally. They vary very much in size and in precise shape, the largest occurring in convolutions of the median parts of the hemispheres.

This gray cortical layer becoming folded through the process of growth, ultimately presents irregular swellings and depressions of its surface. The swellings are called convolutions or gyri; the depressions, fissures or sulci. Some of the sulci are very deep, and receive special names. It should not be forgotten that the bottom of every sulcus is formed by the same ganglionic gray matter as the prominent parts or gyri.

These gyri are so grouped and separated by large sulci, that we are now enabled to make a successful topographical study of the apparently confused mass of convolutions; and in my brief description of the cortex cerebri I shall almost follow Ecker's classification of its parts. Thus in each hemisphere we have four lobes or groups of gyri, viz., the frontal, parietal, temporal, and occipital lobes. Separating these lobes are three large and constant fissures: the fissure of Sylvius, between the frontal and temporal lobes; the fissure of Rolando (or central f.), separating the frontal and parietal lobes; and the occipito-parietal fissure in the inner face of the hemisphere, limiting the occipital and parietal lobes.

Besides, we recognize four lobules, viz., lobulus cen-

tralis (island of Reil at the bottom of the fissure of Sylvius), lobulus paracentralis, lobulus cuneus, and lobulus quadratus, on the inner surface of the hemisphere. The paracentral lobule is made up almost wholly of the upper (inner) ends of the ascending frontal and ascending frontal convolutions as they dip into the great longitudinal fissure.

For a full account of these parts I would refer you to Ecker's monograph on the cerebral convolutions, to Ferrier's work on the functions of the brain, and to the latest edition of "Dalton's Physiology." A few of the convolutions in these lobes and lobules are of importance in the study of localization, and I must briefly describe them.

First, the faini and ascending frontal convolutions. The former of these (f. 3, Fig. 9) constitutes the lower tier of gyri in the external aspect of the frontal lobes, and forms the anterior superior lip of the fissure of Sylvius. Its posterior part and its continuation into the island of Reil certainly have a very close connection with the function of written and spoken speech. The ascending frontal gyrus (A, Fig. 9) forms the posterior limit of the frontal lobe, and lies against the fissure of Rolando. Ecker calls it the anterior central convolution, but in common with Prof. Charcot and his pupils, I prefer the former designation. Immediately behind the fissure of Rolando, extending almost vertically, is the ascending parietal gyrus, or the posterior central convolution of Ecker. These two gyri, the ascending frontal and parietal, are intimately connected with movements of the face, arm, and leg; so-called centres for the face existing in the lower ascending frontal, centres for the upper extremities being found in its middle portion, and in the ascending parietal gyrus; while the centres for the movements of the lower limbs are in the upper (inner) extremities of both these gyri, and in the next parietal convolution near the median fissure, viz., the superior parietal (p. 1, Fig. 9).

The last gyrus of clinical importance is the next below the inferior parietal and its extension toward the occipital lobe, the angular gyrus, so-called (P_2' Fig 9). Some recent clinical and post mortem facts

would seem to connect it with movements of the eyelids and upper face. Besides, the paracentral lobule is of importance to us, because we know that its destruction is followed by descending degeneration.

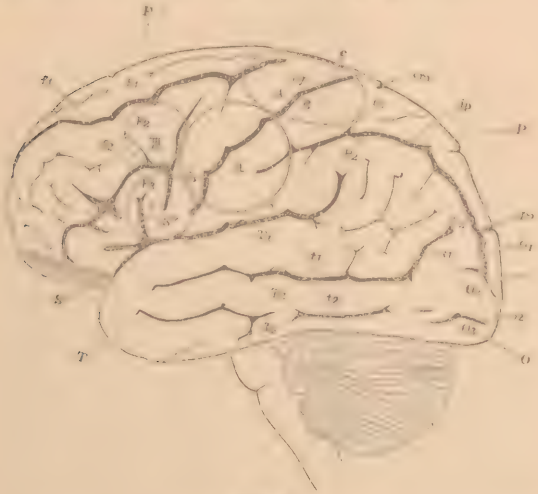

Fig. 9.

Modified from Ferrier: letters and figures the same.—S, Fissure of Sylvius; c, Fissure of Rolando; po, Parieto-occipital fissure. A, Ascending frontal gyrus; B, Ascending parietal gyrus; F_3, Third frontal gyrus; P_3', Gyrus angularis. Circle I., Seat of lesions which (on the left side) cause aphasia. Circle II., Seat of lesions which convulse or paralyze the upper extremity of the opposite side. Dotted Circle III., Seat of lesions which probably convulse or paralyze the face on the opposite side. Dotted Oval IV., Seat of lesions which probably convulse or paralyze the lower extremity of the opposite side. These districts receive their blood-supply chiefly from the middle cerebral artery.

The cerebral convolutions are supplied with blood by branches of the anterior, middle, and posterior cerebral arteries, and an exact knowledge of the distribution of these trunks and their ramification is of the utmost importance in the study of localization of lesions, inasmuch as one of the most common pathological processes by which destruction of convolu-

tions is caused, is embolism, or plugging of one of these arteries.

In general terms, the anterior cerebral artery supplies the inner face of the hemisphere as far back as the occipito-parietal fissure; the first, second, and (very partially) the ascending frontal convolutions.

The middle cerebral artery, or the Sylvian artery, is the most important physiologically, as it supplies all the convolutions mentioned above as concerned in the production of voluntary movements, viz., the third and ascending frontal, ascending, and first and second parietal convolutions. Easily recognized branches of the middle cerebral artery furnish blood to the third frontal (first branch in the fissure of Sylvius), to the ascending frontal, to the ascending parietal; a fourth branch extends as far as the angular gyrus, and a fifth supplies the first temporal convolution.

The posterior cerebral artery supplies the remainder of the temporal lobe and the whole of the occipital.

It might not quite be out of place to state here that, according to Charcot and Duret, the basal ganglia of the brain receive their blood through small branches which leave the great arteries very near the origin of the circle of Willis. The anterior part of the nucleus caudatus is supplied by arterioles derived from the anterior communicating artery, and from the first portion of the anterior cerebral artery. The nucleus lenticularis and the anterior part of the thalamus opticus are vascularized by branches of the trunk of the middle cerebral artery before it enters the Sylvian fissure. The larger part of the optic thalamus is supplied by vessels coming from the second portion of the posterior cerebral artery beyond the circle of Willis. Finally the inner aspects of the thalamus and the walls of the third ventricle receive branches from the posterior communicating artery, and from the first portion of the posterior cerebral artery within the circle of Willis.

To return to the arteries of the cortex. Ramifying in the pia covering the convolutions, they penetrate the nervous tissue in a peculiar manner, in the shape of long and straight branches, which supply the various layers of the cortex by means of horizontal branches, and ultimately, in small numbers, and greatly reduced

in size, reach the white substances. A most important peculiarity in the superficial and deep cortical circulations is the absence of anastomosis between arteries of any great size. As to the exact amount of anastomosis there is a difference of opinion between the two original observers in this matter—both equally competent—Duret, of Paris, and Heubner, of Leipzig. The former maintains, and pathology supports him, that there is next to no anastomotic circulation upon or in the cortex (except through capillaries), while Heubner thinks that considerable branches of the great cerebral vessels open into one another upon the surface of the brain. The importance of this point for the prognosis of embolism of the cerebral arteries is enormous, and for my part I would say that I am disposed to consider Duret's statement as more applicable to practice.

I now pass to a very short account of the physiology of the cortex. My statements upon this matter will be all the briefer because an excellent and full account of the physiology of the cerebrum is accessible to all of you in Prof. Dalton's "Treatise on Physiology." Dr. Dalton himself has taken an honorable part in the researches which have, in the last five years, revealed unsuspected properties in the cortex of the brain.

First. We now know, since the experimental researches of Fritsch and Hitzig (1870), and of Ferrier (1873), that the cortex of the brain is excitable; *i. e.*, that galvanization or faradization of the cortex produces muscular movements in the body and limbs. This fact, standing out in direct contradiction to the teaching of all physiologists from Magendie and Müller, is a monumental acquisition to biological science.

Second. Very numerous researches by Hitzig, Ferrier, and a host of others, appear to have established beyond question that a certain relation exists between well defined portions of the cortex of one hemisphere and limited muscular groups (almost individual muscles) in the opposite half of the body. The areas of convolutions whose irritation by electricity is followed by definite movements of peripheral parts have been denominated motor centres, or psycho-motor centres; and a large number of these centres have been deter-

mined by Prof. Ferrier and by Prof. Hitzig, upon the brains of dogs, cats, and living monkeys whose brains bear a certain resemblance to the cerebrum of man. While knowing of a few opposing experiments which would seem to show that there is no constant relation between the spot irritated and the resultant movement, I am bound, by the weight of evidence and by the wonderful accord between the researches of various experimenters, to accept the facts as stated above.

Third. The excitable district of the brain is its median group of convolutions, including (as sketched upon a human brain, after experiments upon monkeys) the second, third, and ascending frontal convolutions, the ascending and first parietal convolutions. This excitable district or zone includes, as you perceive, gyri which receive their blood-supply by branches of the middle cerebral artery, with the exception of the second frontal gyrus. As my chief object is not physiological teaching, I prefer simply to enumerate the cortical centres as laid down by Ferrier, not encumbering the diagram with a representation of them.

On the posterior extremity of the third frontal gyrus, near the fissure of Sylvius, is a centre for the movements of the lips and tongue (a speech centre according to the teaching of pathology); this is numbered 9 and 10 on Ferrier's plate. Next in order, upon the lower part of the ascending frontal convolution are centres for movements of the elevators and depressors of the angle of the mouth; numbered 8 and 7. Still higher on this gyrus is a centre for movements of the forearm and hand; numbered 6. Upon the upper two-thirds of the ascending parietal convolution are several centres for complex movements of the head and wrist; designated by Ferrier, *a*, *b*, *c*, *d*. Much farther forward, upon the hemisphere near the great longitudinal fissure, is an extensive region embracing the posterior parts of the first and second frontal gyri, governing lateral movements of the head, elevation of the eyelids, and dilatation of the pupil; numbered 12. Immediately behind this, near the longitudinal fissure, upon the posterior extremity of the first frontal convolution, is a centre for extension and

forward movements of the hand and arm; numbered 5. The posterior (inner) ends of the ascending frontal and ascending parietal convolutions contain centres (not clearly differentiated) for complex movements of the arms and legs together; numbered 2, 3, and 4 on Ferrier's plate. Finally, behind these on the superior parietal lobule is a centre for movements of the leg and foot; numbered 1.

The sensory centres of Ferrier occupy various parts of the inferior parietal lobule, the gyrus angularis, the second occipital, and first temporal convolutions. Although the experiments of various observers make it exceedingly probable that these inferior and posterior portions of the hemisphere are connected with general and special sensory functions, yet, as human pathology has so far thrown no light upon these questions, I shall hereafter confine myself to the study of the motor districts of the convolutions.

Not to weary you by the citation of the now very numerous cases in which localized cortical lesions, as tumors, abscesses, clots, softening, pressure effects from bone or thickened meninges, have given rise to definite symptoms, in close or even almost exact agreement with the data obtained by faradizing the cortex, I shall state in a general manner the tendency of these recent clinical and post-mortem studies.

In the first place, it appears almost absolutely certain that in man a lesion involving the posterior part of the third frontal convolution (on the left side usually) causes aphasia; *i. e.*, impairment or loss of articulate speech, or even of language in general. It would seem, besides, that (1) lesions of the same part on either side of the brain produce paresis of many muscles concerned in lingual and pharyngeal movements; (2) that lesions of the anterior folds of the island of Reil, convolutions which are continuous with the third frontal, may also produce aphasia; and that (3) loss of speech may result from injury to the white substance lying between the third frontal gyrus and the basis cerebri. I believe, you observe, in a not too limited localization of the motor functions exerted in language, and would graphically represent this by the circle marked I. in Fig. 9.

In the second place, lesions limited to the inferior portions of the ascending frontal and parietal gyri have produced spasmodic and paralytic phenomena limited to the upper extremity of the opposite side. I am disposed to admit as highly probable that these parts are connected in the healthy living man with the various volantary movements of the arm and hand. This zone is represented in Fig. 9 by circle II.

I am not prepared to go farther in admitting pathologically proved cortical centres, but would add that there are some reasons for believing that future autopsies will locate one centre for the external facial muscles just forward of the two centres named above, viz., the region included in the dotted circle III.; and another for movements of the legs upon the upper parts of the ascending frontal and parietal, as roughly indicated by dotted oval IV.

As regards sensory cortical centres, I have already said that we have as yet no pathological data for their study.

Having thus expressed myself about the question of cortical centres in man, I pass to the more clinical study of symptoms observed when the cortex is injured.

First. What are the symptoms of localized lesions involving the cortex alone, or the cortex and a minimum of subjacent white matter? The symptoms differ vastly in accordance with a rule laid down years ago by Brown-Séquard, according as the lesion is an irritative or destructive one: and besides, they vary according as the lesions are within the excitable cortical region (defined *supra*) or outside of it. We can clear the ground pretty safely at once by admitting that lesions irritating or destroying convolutions not embraced in the motor zone produce no symptoms at all. Large parts of the frontal, temporal, or occipital convolutions may be injured or utterly destroyed without the patient showing during life any special symptoms of organic cerebral disease. This statement is based upon the study of recent cases only, though I doubt not that in the literature of the century numerous apparently contradictory cases might be collected. In considering this negative

proposition, one proviso must be borne in mind, viz., that if the lesions of these unexcitable districts involve the dura mater, convulsions and localized cephalalgia may occur. Of this I have seen one marked example. We are now prepared to study the symptoms of lesions in the excitable or motor zone of the hemispheres, as indicated by the various circles in Fig. 9.

1. The symptoms of an irritative lesion of these parts consist in convulsions, with or without subsequent transient paralysis; *e. g.*, such a lesion in circle III. (Hitzig's case) would give rise to spasmodic movements in the superficial muscles of the face on the opposite side, with slight paralysis. Irritative lesions of the regions included in circles II. and IV. will cause convulsions limited to, or first appearing in the hand and arm, or foot and leg, of the opposite sides. As regards circle I., Broca's speech centre, we know little of the effects of its pathological irritation. In one case which I have placed on record, a thickening of the meninges involving the third frontal convolution of the left side produced intermittent and incomplete aphasia.

It was by the close study of the clinical and pathological aspects of cases of localized epilepsy (fingers and hands), that Dr. J. Hughlings Jackson was enabled to form his theory of motorial discharges from irritation of the cortex cerebri, and thus pave the way for Ferrier's admirable researches. Dr. Jackson must, I think, be considered, after Prof. Broca, as the founder of our present growing doctrine of cortical localizations.

2. Destructive lesions of portions of the excitable district produce paralysis in peripheral parts across the median line. The symptoms will, to a certain extent, correspond with the *precise* location of the lesions, very much as in irritative lesions; *e. g.*, embolism of the first branch of the middle cerebral artery on the left side will cause softening of the posterior part of the third frontal gyrus, with the symptom aphasia. A destructive lesion of the principal part of the motor zone on the right side will produce left hemiplegia without aphasia; but if this lesion occupy

the left hemi-sphere, loss of speech will co-exist with the paralysis.

It must be added that secondary descending degeneration ensues after destructive lesions of the motor regions of the cortex, and that we have late contracture or rigidity of the paralyzed limbs as part of the symptom-group.

Negative characters of these cortical lesions are preservation of sensibility in the paralyzed parts, and (except with epileptic attacks) preservation of consciousness, and incompleteness of paralysis.

In the next place, let us inquire what are the symptoms produced by diffused lesions of the cortex. As exemplified in acute meningitis, the chief symptoms are delirium, convulsions, and pain; evidences of intense irritation. The coma and paralysis which follow may in some degree be caused by impaired nutrition of the cortex, but more probably by circulatory and tension-changes in the whole encephalic mass.

There is a much better disease for studying the effects of lesions of the surface of the brain, half irritative and destructive—I mean general paralysis of the insane, or, anatomically speaking, diffused chronic meningo-encephalitis. The affection is very common, and has been thoroughly studied clinically and pathologically. From these studies we learn that in the first stage of the affection there occur fibrillary contractions in many muscles of the tongue, face, and limbs; that speech is made tremulous and jerky; that there is over-talkativeness and even acute delirium; that gradually memory and judgment become impaired, and a semi-paralytic and semi-ataxic condition develops in the limbs. Later the mental faculties are abolished; a stage of dementia with occasional gleams of delusions (exalted notions), and integrity of the organic functions characterize the disease. The attempt is now being made to show that when the meningo-cortical changes are limited to the frontal lobes the symptoms are mainly psychical; when the lesion involves the motor districts alone we observe abundant fibrillary tremors and pseudo-paralysis; and finally, if the occipital lobes are affected, sensory symptoms (hallucinations) predominate. As yet not much support has been ob-

tained for such a distinction, which appears very tempting upon physiological grounds. It should not be forgotten, in using cases of general paralysis for the study of the question of localization, that the disease is one in which lesions exist in many parts, or almost all the parts of the cerebro-spinal axis.

The question of the localization of functions in the cerebral convolutions, and that of the possibility of diagnosticating their lesions are as yet in their infancy: we need numbers of exact observations to decide it one way or the other. Just now, I believe that the presumption is in favor of a positive answer: there are many facts supporting this affirmative. The clinical and post-mortem facts have just been referred to, and I shall close the lecture by recapitulating the various anatomical, physiological, and pathological evidence in favor of the existence of motor centres in the cortex.

1. Coarse anatomy enables us to trace bundles of fibres upward from the motor tract of the medulla and pons, into the internal capsule as far as the convolutions which are grouped about the fissure of Rolando. By its aid we can also trace sensory nerve-fasciculi from the posterior regions of the pons to the occipital and temporal lobes, and their cortex. Such gross dissections are, however, condemned as unreliable by some authorities.

Microscopic anatomy shows that the so-called motor gyri are rich in large cells; nay, that they alone contain the "giant-cells" of Betz, that is, ganglion-cells, which in size and number of processes bear a remarkable resemblance to the unquestionably motor ganglion-cells of the anterior horns of the spinal cord and the medulla oblongata. In the motor convolutions these giant-cells are found in little clusters of three, five, or more, in a section, imbedded among the large ganglion-cells of the third layer.

2. Experimental physiology teaches us that electrical irritation of this zone, and of this zone only, produces muscular contraction in parts on the other side of the median line; and, further, that this zone may be divided into a number of "centres" for various small

parts, tongue, face, arm, leg, etc.* By experimentation we also learn that shining or burning off these cortical centres produces partial paralysis of peripheral parts on the opposite side of the body, with precisely the same correspondence between centres and muscular groups as the irritative experiments demonstrate.

By the latter mode of experimenting applied to the occipital and temporal convolutions (Ferrier, H. Munk, and others), it is made highly probable that there is a certain relation between parts of these gyri and the organs of special and general sensibility across the median line.

3. Pathological anatomy (recent cases) demonstrates (*a*) that destructive lesions of the motor regions of the cortex (and of the paracentral lobule) produce descending degeneration throughout the direct cerebral motor tract extending into the lateral columns of the spinal cord; and (*b*) that there is a remarkable correspondence between certain localized spasmodic and paralytic symptoms observed during life, and lesions irritating or destroying certain definite spots in the motor zone of the cortex.

Lecture VIII.

SUMMARY:—SURGICAL ASPECTS OF THE QUESTION OF CEREBRAL LOCALIZATIONS—CRANIO-CEREBRAL TOPOGRAPHY, AND ITS UTILIZATION IN DIAGNOSIS AND FOR OPERATIVE PROCEDURES.

GENTLEMEN:—The question of the utilization of the doctrine of localization in surgery requires to be studied. This, the most novel part of the subject, is, I think, of great present interest and of much promise in the future. Already several brilliant surgical operations have been performed upon indications derived from

* Still more recent researches by MM. Franck and Pitrès show that after removal of the cortex in the excitable zone, faradization of those portions of white substances which are then exposed (anterior half of the internal capsule) gives rise to similar (in kind and in distribution) movements in peripheral parts across the median line.

the newly acquired knowledge of cranio-cerebral topography. By this term we mean the determination of the relations between the external surface of the skull and the principal gyri and sulci of the brain. So little was done toward ascertaining these relations that up to 1861 the position of the fissure of Rolando relative to the coronal suture was wholly unknown. In that year Prof. Broca invented a scientific procedure for the study of the subject: he inserted pegs into the cerebral substance through holes drilled into the skull at given points, and then, removing the skull-cap carefully, was enabled to determine exactly what convolutions corresponded to the pierced regions of the skull. He thus discovered that the parieto-occipital fissure lies under the lambdoidal suture, and that the fissure of Rolando slopes backward, so that its posterior extremity is placed at more than forty millimetres behind the coronal suture. Since that year the subject has been thoroughly studied by Broca, Rischoff, Hüttler (1873), Prof. Turner, of Edinburgh (1874), Ch. Féré and Broca (1875), and others. Perhaps the best and most applicable of these contributions is that of Féré, and I shall follow it closely in the following remarks. It should be borne in mind that for purely anthropological purposes the determination of the relation of gyri and sulci to certain sutures or processes of the bare skull is sufficient; but that for use in the regional diagnosis of cerebral injuries, and in practical surgery, the cranial landmarks should be such as are easily determined upon the scalp and face of the living man. The *system* of cranio-cerebral topography which I offer for your guidance is based upon the latter principle of study.

As shown in Fig. 10, I shall sketch the situation of the principal convolutions, fissures, and central gray bodies of the cerebrum upon an outline figure of the profile of a skull. The skull is represented as resting upon a peculiar plane, one passing under the condyles of the occipital bones and the alveolar processes of the superior maxillæ—the alveolo-condyloid plane of Broca. Upon this horizontal line, which can be determined with reasonable accuracy in the living human being, we erect other lines and measure dis-

tances which enable us to solve almost the whole problem.

1. A vertical line (A) drawn from the alveolo-condyloid plane through the external auditory meatus upward will pass through or very near to the bregma or line of junction of the frontal and parietal bones at the vertex; it passes through the anterior (lower) extremity of the fissure of Rolando.

2. If from the upper end of this vertical line A, we measure a distance of 45 mm. backward toward the occiput and draw a descending vertical line (1–2), we mark out the location of two most important parts of the cerebrum, viz., the posterior extremity of the fissure of Rolando, and the posterior limit of the thalamus opticus in the hemisphere [at *c*)].

3. To conclude with the occipital end of the skull; if we can make out with the finger the lambdoid suture at the median line, we thus learn the situation of the subjacent occipito-parietal suture, which separates the parietal and occipital lobes.

4. The last vertical line worth noting is one drawn at a distance of 30 mm. forward of the auriculo-bregmatic line. This vertical line (3–4) will pass through the middle fold of the third frontal convolution (just forward of the speech-centre), and will also indicate the anterior limit of the central cerebral ganglia, viz., the head of the nucleus caudatus in the hemisphere [at (*d*].

5. The upper level of the central cerebral ganglia may be quite exactly indicated by a horizontal line drawn at a distance of 45 mm. below the surface of the scalp at the bregma (or 35 below the surface of the bare skull at the same point). This line (7–8) also extends across the middle regions of the motor district of the convolutions, containing centres for the face and upper extremities.

6. The external angular process of the frontal bone, not difficult to define in the living subject, is the starting-point of another horizontal line (5–6), whose posterior extremity passes a little below the lambdoid suture. Upon this horizontal line we can, by measurement, determine the location of certain parts. Thus, at a distance of 18 or 20 mm. behind the external

angular process (the folded part of the third frontal convolution (a). This point in many heads will correspond with the vertical line 3, 4.

FIG. 10.—Outline of skull resting upon the alveolo-condyloid plane of Broca; modified from Topinard (*Anthropology*). Vertical line A, or auriculo-bregmatic. Line 9–10 drawn parallel to the plane of Broca. Upon this line, at a distance of 45 mm. posterior to the bregma, a vertical line, 1–2, will pass through the upper (inner) end of the fissure of Rolando, *b b*, and through the posterior extremity of the thalamus opticus, *c*). A third vertical line, 3–4, drawn at 30 mm. forward of the bregma, will pass through the fold of the third frontal gyrus, *a*, and through the head of the nucleus caudatus (*d*. The horizontal line 7–8, at 45 mm. below the bregma (scalp), indicates the upper limit of the central ganglia. The third horizontal line 5–6, passing through the external angular process of the frontal bone and the occipito-parietal junction, approximately indicates the course of the fissure of Sylvius, and serves for measurements. At 18 or 20 mm. behind the ext. ang. process on this line is the speech-centre of Broca; 5 to 8 mm. behind the intersection of 3–4 and 5–6, is the beginning of the fissure of Sylvius, and at 28 or 30 mm. behind this intersection is the lower end of the fissure of Rolando, *b*, *b*, placed a little too far back in the cut. At × (near 6), near the median line, is the location of the occipito-parietal fissure.

7. The situation of the fissure of Sylvius may be approximately ascertained in the following manner: Its middle portion extends horizontally, almost under the

upper part of the squamous suture, which in the living subject is to be found a little below the horizontal line 5–6. The anterior extremity or beginning of the fissure of Sylvius is a little below this horizontal line, at a distance of some 5 to 8 mm. posterior to the intersection of 3–4 and 5–6, and consequently about 22 or 25 mm. anterior to the auriculo-bregmatic line A. Lastly, according to Turner, the parietal eminence almost always overlies the supramarginal gyrus (P, Fig. 9), consequently the posterior extremity of the fissure of Sylvius is likewise in this vicinity.

8. The angular gyrus is to be found below and behind the parietal eminence, a little above the horizontal line drawn from the external angular process (5–6).

9. The anterior (lower) end of the fissure of Rolando lies at a distance of 28 or 30 mm. behind the line 3–4, and a little above 5–6. It is therefore a few mm. anterior to the vertical line A.

The application of these data to practical medicine and surgery is quite obvious.

In medical cases, when tumors of the skull develop externally, we may determine by cranio-cerebral topography whether their extension inward can give rise to the motor symptoms which are present (convulsions and paralysis), according as the cortical district of the brain are threatened by extension of the growth from the inner surface of the skull, or not.

In surgery, the utility of cranio-cerebral topography is much greater.

For example, as far back as 1871, Professor Broca was able to correctly diagnosticate an abscess of the left third frontal convolution, and was successful in trephining directly over it. In 1876, Pitres and Lucas-Championnière successfully trephined the skull in two cases, for the removal of fragments which were compressing the ascending frontal and parietal convolutions, and causing paralysis.

In the case of a patient who is paralyzed on one side of the body, after an injury to the skull, the following considerations might justify, or even indicate an operation for the removal of bone, of blood, or of pus:

1. If the hemiplegia (or hemi-spasm) be very complete, it is probable that the injury to the brain is considerable in extent, and extends deeper than the special centres for the face, arm, and leg.

2. If the externally evident injury be over non-excitable convolutions, and the paralytic or spasmodic phenomena be marked, it is more than probable that the brain is torn or compressed at other points than under the seat of injury, and an operation is contra-indicated.

3. If the paralytic or convulsive symptoms be on the same side as the evident cranial injury, it is probable that there are cerebral lesions on the other side produced by *contre-coup*, hence interference will be undesirable.

4. Even if a cranial injury be directly over excitable convolutions, if the resulting paralysis or convulsions be accompanied by marked anæsthesia, an operation cannot be expected to do much good, because the presence of anæsthesia makes it highly probable that the white substance of the hemisphere (posterior half or third of the internal capsule) is involved in the lesion.

5. A favorable indication for trephining is when aphasia supervenes immediately, or in a few weeks after an injury to the skull in the region of the left third frontal convolution (see Fig. 10). It is extremely likely, in the first case, that a clot or spiculum of bone will be found compressing or lacerating the centre for speech; in the second, that an abscess has formed in the same part (Broca's case).

6. Another combination of symptoms which makes an operation desirable, and holds out a hope of its being successful, is when an injury to the skull over the fissure of Rolando on one side is accompanied by slight hemiplegia, or by paralysis of the face, or arm, or leg, or of any two of these parts combined, on the opposite side of the median line, without anæsthesia. Under such circumstances, the probabilities almost amount to certainty that the centres for (see Fig. 9) the face, arm, and leg are separately or collectively involved in the lesion (cases of Proust and Lucas-Championnière).

7. A contra-indication to operative interference, even in apparently favorable cases, would be symptoms of basal lesions, such as palsy of cranial nerves, neuro-retinitis, Cheyne-Stoke's respiration, and vomiting.

Addendum.—A number of corrections to the propositions in these lectures have been suggested by my friends, or have occurred to my own mind, but I believe that only one is worth inserting here. The other corrections and additions are right enough in a theoretical sense, but as I have tried to adhere to my rule of making no statement which does not receive support from several sources, normal anatomy, pathological anatomy, and clinical observation, I prefer not to publish them.

The correction which I wish to make is to the paragraphs *a*, *b*, and *c*, in Lect. V., p. 103, descriptive of the crossed paralysis caused by lesions of the crura. It should be added in each of these paragraphs that the lower part of the face is usually paralyzed on the same side as the body palsy, *i.e.*, on the side opposite the lesion. In case of a lesion involving both crura, the two halves of the face will be paretic.